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No. 94

NEONATAL MORTALITY
AND MORBIDITY

Report by

*A Joint Committee of the Royal College of Obstetricians
and Gynaecologists and the British Paediatric Association*

LONDON: HIS MAJESTY'S STATIONERY OFFICE

1949

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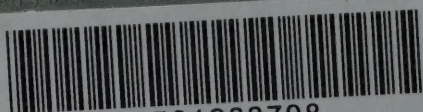
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PREFATORY NOTE

by the Chief Medical Officer

To the RT. HON. ANEURIN BEVAN, M.P.,
Minister of Health.

SIR,

I have the honour to present a Report on Neonatal Mortality and Morbidity prepared by a Joint Committee of the Royal College of Obstetricians and Gynaecologists and the British Paediatric Association.

In 1942 the British Paediatric Association, reacting vigorously to a criticism that its members were more interested in curative than in preventive medicine, began a study of the problem of neonatal mortality. The conclusion was soon reached that such a study would more profitably be undertaken in collaboration with the Royal College of Obstetricians and Gynaecologists. As a result a Joint Committee of the College and the Association was constituted as follows:

Royal College of Obstetricians and Gynaecologists.

Sir Eardley Holland (*Chairman*).
Mr. A. C. H. Bell.
Professor F. J. Browne.
Professor A. M. Claye.
Dr. E. C. Fahmy.
Professor T. N. A. Jeffcoate.
Professor W. C. W. Nixon.

British Paediatric Association.

Professor Sir Leonard Parsons (*Deputy Chairman*).
Dr. F. M. B. Allen.
Professor Norman Capon.
Dr. W. R. F. Collis.
Dr. A. White Franklin.
Professor W. F. Gaisford.
Professor Stanley Graham.
Dr. Reginald Lightwood.
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Professor Alan Moncrieff.
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After the preliminary work had been done, Professor Capon and Professor Jeffcoate drafted the report in its present form. Before publication it was approved by the Executive Committee of the British Paediatric Association and the Council of the Royal College of Obstetricians and Gynaecologists.

It is appropriate that an important study of this nature should be published as one of the series of official reports of the Ministry of Health on Public Health and Medical Subjects.

I have the honour to be, Sir,

Your obedient Servant,

WILSON JAMESON

WHITEHALL

January, 1949.

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I. PRELIMINARY CONSIDERATIONS

ALTHOUGH it is with the first month of infant life that this report is chiefly concerned, a broader view must be taken if the various problems are to be seen in proper perspective. The neonatal period must be considered in relation to the developmental stages by which it is preceded.

Life starts when the ovum is fertilized and then proceeds through an intra-uterine period of progressive foetal development to the expulsion at birth of an infant fully prepared for independent existence. Any influence which adversely affects this foetal development will be reflected in the state of the infant at birth. It is necessary, therefore, to consider the main influences bearing on the early phases of human life.

Already at conception the forces of heredity are in action for good or ill. The part played by hereditary influences in the causation of neonatal disease and death is not yet clearly defined. Diseases such as epilepsy, amaurotic family idiocy and acholuric jaundice are usually not manifest until after the newborn period, although they are undoubtedly hereditary. Haemolytic disease of the newborn is of such special importance that it is considered separately (see Chapter IX). According to Haldane (1944) it is responsible for more stillbirths and neonatal deaths than any other inherited condition. Congenital syphilis, although inherited, is not of genetic origin, being due to the direct passage of the infecting organism through the placenta into the foetal circulation. Certain types of foetal malformation (see Chapter V) appear to be hereditary, the familial incidence of harelip and cleft palate, for example, being noteworthy. Gross deformities such as anencephalus and spina bifida may also be hereditary but direct evidence on this subject is difficult to obtain and the chance of affected infants surviving to beget offspring is remote. There is scope for further research here. Until the influence of heredity is more clearly understood the contribution which eugenics may make to neonatal health cannot be assessed.

Immediately after conception, alterations in biophysical or biochemical activities in the maternal organism may adversely affect the implantation of the ovum and profoundly upset the future development of the foetus, causing miscarriage, stillbirth or congenital abnormality. Once the ovum is healthily implanted, the growing embryo continues to depend on the maternal organism for protection and nutrition and it is clear that the mother's physical and mental state and her environment, largely directed by economic and social status, play an integral part.

The human foetus takes approximately thirty-eight weeks to develop to maturity and it is of the utmost importance that this full period of intrauterine life should be completed. Premature birth constitutes one of the major factors in neonatal mortality. The birth-process, either at term or before, always involves the foetus in mechanical stresses which are potentially dangerous and which may result in intracranial damage or asphyxia.

Directly after birth comes the neonatal period with its problems associated with circulation, respiration, temperature control, digestion, etc., and the risk of exposure to infections against which there is but little natural immunity.

Chronologically the chief causes of neonatal mortality and morbidity are congenital malformations, prematurity, birth injury, asphyxia and infections.

Probably 60 to 70 per cent. of neonatal deaths are due to conditions related to the birth—prematurity, injury or asphyxia. In many cases there may be more than one possible cause of death and it is imperative that, whenever possible, the primary or dominating cause should be discovered. This means that post-mortem examinations are essential in all cases of stillbirth and neonatal death if prophylactic measures are to be improved.

STATISTICAL EVIDENCE

Neonatal mortality and morbidity must be considered in conjunction with stillbirths. The difference between a stillbirth and a neonatal death* is often very slight and both have many causes in common. Moreover, many infants registered as stillbirths on the Continent of Europe (where pulmonary respiration is accepted as the sign of live birth) would be considered as neonatal deaths in Great Britain (where the presence of a heart-beat is regarded as the sign of live birth). Consequently the stillbirth rate on the Continent is relatively higher than in Great Britain and caution must be exercised in making comparisons between the statistics of different countries. Again, it is likely that the definition of stillbirth is not always adhered to in practice and doctors in this country may occasionally report as stillborn, not only babies born dead *before* the 28th week of pregnancy, but viable babies whose hearts were beating at birth, and even some of those who made feeble unsuccessful attempts at respiration. Another factor which influences the reliability of statistical evidence is the difficulty of making an accurate diagnosis of the cause of neonatal death, especially without a post mortem examination. Birth trauma or neonatal infection, for example, may not appear on the death certificate of a premature baby, "prematurity" being given as the cause of death, although either trauma or infection may have been the real determining factor. Similarly, "congenital debility" or some such term may conceal the fact that atelectasis or inadequate nursing care or sepsis was primarily responsible for death.

There are, nevertheless, some points of importance that may be learned from a study of statistics. These were set out in a report "Infant Mortality in Scotland," by the Department of Health for Scotland (1943), and there is no need to repeat the figures in full. As far as England and Wales are concerned the stillbirth and neonatal statistics may be summarised as follows. The stillbirth rate (i.e., the number of stillbirths registered during the year per 1,000 live and stillbirths registered during the same year) from 1931 to 1935 was 41. In 1944 and 1945 it had fallen to 28. Different areas show varying rates ranging (1939 to 1941) from 30 in Greater London, to 43 in Manchester and Salford, and 46 in Wales. There is evidence that the rate is higher among the working class population than among the upper middle and professional classes (see Chapter IV). The total number of stillbirths registered during 1945 was 19,333 and the number of live births was 679,937.

The infant mortality rate in 1945 (deaths occurring within one year of birth) was 47 per 1,000 live births and of these 24.8 per 1,000 died during the neonatal period. The total number of infant deaths was 31,959 and there were 16,910 neonatal deaths—that is, more than half of all infants dying in England and Wales during the first year of life died during the neonatal period.

* Definitions in Great Britain:

Stillbirth—any child which has issued forth from its mother after the 28th week of pregnancy and which did not at any time after being completely expelled from its mother, breathe or show any other signs of life.

Neonatal death—death occurring within the first four weeks after birth, i.e., during newly born or neonatal period.

When these figures are compared with those of the five year period, 1906-1910, during which the infant mortality rate was 117 and the neonatal death rate 40, it will be seen that the saving of life during the last eleven months of the first year of life has been much more significant than during the first month.

In New Zealand the neonatal mortality rate from 1936 to 1940 was 23, and in 1943 it was 21. In Holland (1936-1939) it was 21 and in Amsterdam in 1937 it was as low as 16. Greater London and South East England have rates which will compare with those of the best countries, but some of our cities have distressingly high rates, e.g. (1934-1938) Newcastle and Gateshead 41, Stoke-on-Trent 37, Sunderland 36. During this time London's figure was 24 and Croydon 22. As an example of a recent figure in a large industrial area, Liverpool's was 29.8 in 1945. The neonatal mortality rate, like the stillbirth rate, is considerably higher among the lower income group of the population than amongst the well-to-do (see Chapter IV).

During 1945 more than 36,000 women in England and Wales suffered the inconveniences, dangers and disappointments of fruitless pregnancy—and this takes no account of the loss of potential infant life by abortion. Titmuss (1944) estimated that out of nearly two million pregnancies in the three-year period 1936-1938, more than a quarter of a million, or 1 in 8, children were lost as a result of spontaneous abortion, stillbirth, or death before the age of one year.

If the present stillbirth and neonatal death rates in England and Wales were reduced to those obtaining in Holland in 1938 (25 and 21 respectively) there would be a saving of approximately 4,000 lives annually. To be content with this, however, would show unjustifiable complacency because there is now evidence to show that considerably better figures can be obtained. Baird (1945) reported two series of well-to-do patients in which the neonatal death rates were only 8.1 and 13.7, and Allen, Macafee and Biggart (1943) recorded a rate of 9.4. Moreover it is likely that most obstetricians in this country could show similar figures for personally treated cases taken from the higher income groups (private patients). The Report of the Chief Medical Officer of the Ministry of Health for 1939 to 1945 also provides significant evidence. During these war years 152,000 women, selected as normal cases, were delivered in emergency maternity homes in reception areas, with a neonatal mortality rate of only 9.6 (maternal mortality rate 0.8 per 1,000 births). Thirty thousand confinements took place in maternity homes upgraded to admit abnormal cases and the neonatal mortality rate was 13.9 (maternal mortality rate 1.9 per 1,000 births). Although the number and severity of obstetric abnormalities in this latter series was probably not comparable with those dealt with in the larger teaching maternity hospitals, they may be considered roughly representative of those encountered in obstetric practice throughout the country as a whole.

It is thus apparent that a more general application of present day knowledge of obstetrics and paediatrics by the medical and nursing professions and by those responsible for social services and for administering hospital and domiciliary midwifery services would result in a further considerable reduction in the neonatal mortality rate. Similarly, if every pregnant woman received first class medical and nursing supervision during pregnancy and labour, the stillbirth rate could also be appreciably decreased.

The evidence goes to show that the present neonatal mortality and stillbirth rates could be reduced by one third to one half, and if they were, there would be a saving of about 15,000 babies each year in England and Wales alone.

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The statistics for England and Wales in 1945 mentioned in the text have been kindly supplied by the Registrar-General to whom we are indebted for permission to quote them.

II. CONDITIONS PERTAINING TO THE PLACENTA AND MEMBRANES

(a) IMPLANTATION OF THE OVUM

About one week after fertilization the ovum becomes implanted in the endometrium and if normal foetal development is to occur this process must be anatomically and physiologically sound. If the embedding be faulty, abortion may result or the foetus may be poorly developed or even maldeveloped. Although the mechanism of implantation and placental formation is known, the controlling factors are largely hypothetical. It may be supposed that disease of the endometrium or of the other components of the uterine wall will affect implantation adversely and examples of this undoubtedly occur (e.g., fibroids in the uterine wall) but they are rare. Nevertheless, "endometritis" and allied diseases are no longer acceptable as important causes of abnormal placental formation. A disturbance in the preparation of the endometrial bed by the hormones of the ovary might operate, although this still awaits proof. Again, disease of the Fallopian tube, such as results from infection, may prevent the developing ovum from reaching the uterus and implantation may then occur in the tube; in that case the foetus almost invariably dies, and the life of the mother is threatened. Successful implantation probably depends as much upon the health of the early ovum as upon the healthiness and proper functioning of the mother's organs.

(b) POSITION OF THE PLACENTA IN THE UTERUS—PLACENTA PRAEVIA

In placenta praevia the placenta develops in the lower segment of the uterus, and it inevitably becomes partially detached from the wall of the uterus during the later weeks of pregnancy or during labour, and haemorrhage results. The incidence of placenta praevia in hospital practice varies between 1 in 60 and 1 in 100 cases, and is thought to be relatively high in women who have had many pregnancies at short intervals. Its cause is unknown and though disease of the endometrium is sometimes blamed, low implantation of the placenta appears to follow the laws of chance rather than result from disease or circumstances which might be preventable. It is dangerous to both mother and child; until recent years—and even now in some hospitals—about 50 per cent. of babies in these circumstances were born dead or died shortly after birth (Browne (1944) and Berkeley (1936)). The chief dangers to the child are premature birth, asphyxia, and injury resulting from the treatment adopted in the interests of the mother. The risks have been considerably reduced in recent years by improvements in obstetric practice, such as earlier diagnosis; deferring the baby's birth until as late in pregnancy as possible; and the increased use of Caesarean section and blood transfusion. Even so it has been estimated that the total foetal mortality cannot be reduced much below 25 per cent. (Browne (1944)). The risks to the baby are borne out by the following statistics taken from the reports of the Liverpool Maternity Hospital for 14 successive years. The foetal mortality is uncorrected, i.e., gross malformations incompatible with life are included, but only five such cases occurred in the whole series.

Year	No. of Cases of Placenta Praevia	Stillbirths	Neonatal Deaths	Total Foetal Deaths	Maternal Deaths
1932	44	19	4	23 (51.1%)	2
1933	46	14	5	19 (41.3%)	2
1934	47	10	8	18 (37.5%)	1
1935	41	8	7	15 (36.5%)	1
1936	45	6	7	13 (29.5%)	1
1937	68	10	4	14 (20.3%)	1
1938	36	3	7	10 (27.8%)	2
1939	36	5	5	10 (27.8%)	0
1940	26	7	3	10 (39.5%)	0
1941	16	4	0	4 (25.0%)	0
1942	21	4	5	9 (43.0%)	0
1943	27	0	6	6 (21.4%)	1
1944	21	1	2	3 (14.3%)	0
1945	24	1	1	2 (8.3%)	0

The table shows the trend towards improvement both in foetal and maternal mortality rates.

During the first seven of the years quoted there were 112 stillbirths and neonatal deaths out of 327 cases (34 per cent.) whereas during the last 7 years 44 babies were lost out of 171 cases—a foetal mortality* of 25.7 per cent. Daichman and Pomerance (1947) writing recently from America reported a series of 165 with a foetal mortality of 25.8 per cent. and a maternal mortality of 0.6 per cent. There is, however, good reason to believe that the foetal loss could be reduced below 25 per cent. by modifying obstetrical treatment alone. Thus in 1946 Macafee reported a series of 191 cases treated in the Royal Maternity Hospital, Belfast, with only 1 maternal death and 42 foetal deaths (gross foetal mortality rate 22 per cent., corrected foetal mortality 18.8 per

* Including stillbirths and neonatal deaths.

cent.),* whereas in an earlier series of 76 cases treated in the same hospital the foetal mortality was 51.3 per cent. Moreover Macafee's figures suggest that the prospects for the baby can be improved still further. Nevertheless there are few hospitals in the country whose results will as yet bear comparison with the above; indeed, recent reports (Phillips 1946) (Barnes 1946) from two large hospitals still show a foetal mortality of almost 50 per cent.

(c) DISEASE OF THE PLACENTA AND MEMBRANES

Any disease of the placenta or membranes threatens the foetus with asphyxia or malnutrition. Infarction, however, is the only disease which is common and it mainly occurs when the mother is suffering from toxæmia of pregnancy or some general disease such as chronic nephritis. With the formation of successive infarcts more and more placental tissue is put out of action, the foetus tends to become asphyxiated, its development is affected and it is often small and weak at birth. It may be born prematurely or may die before or during birth. A closely allied but more acute condition is seen in accidental haemorrhage (ablatio placentae) when a large area of placenta may be separated from the uterine wall by sudden and massive haemorrhage. This again is often associated with toxæmia of pregnancy and in one type (concealed haemorrhage) it almost always causes the death of the foetus, irrespective of the method of treatment used. This more serious type—serious for the mother as well as the foetus—occurs especially in older women who have had many pregnancies. Although the standard of treatment naturally has some influence, the chief hope of reducing the loss of infant life as a result of these conditions appears to lie in the prevention and early recognition and treatment of toxæmia and in the regulation of the number of infants born to any one woman.

(d) TRANSMISSION OF NOXIOUS AGENTS BY PLACENTA

One of the functions of the placenta is to protect the foetus from any injurious agent circulating in the mother's blood, but the barrier is occasionally penetrated. Usually the baby is protected from any infectious disease that the mother may acquire, but exceptionally virulent organisms and some viruses, e.g., rubella, may reach the foetus. Again, when the mother is actively syphilitic the placenta may itself become infected and the organisms may ultimately pass through it to the foetal circulation (see Chapter IX). Also, both antigen and antibody of the Rh factor (see Chapter IX) may be transmitted through the placenta. In general the substances which pass most easily from mother to child are those which have a simple chemical nature, such as drugs. Lead, quinine and other preparations which are sometimes taken in the hope of inducing abortion and labour may injure the baby either directly or by their effect on the placenta. The use of quinine to induce labour is now generally recognised as being harmful to the baby and there is much to be said for abandoning it.

Anaesthetics and analgesics administered to the mother in labour

From the practical standpoint the most important group of drugs to be considered are the anaesthetics and analgesics administered to the mother during labour, for the relief of pain and distress. A large and increasing number of obstetricians regard the use of these agents in labour as desirable, not only on humanitarian grounds but also with a view to preventing post-confinement neurosis and psychosis. There is good reason to believe that analgesics and hypnotics, when properly employed, may facilitate the progress

* Corrected by elimination of those cases in which death was due to coincidental conditions such as foetal malformation, gastro-enteritis, etc.

of natural labour by relieving pain and allaying the mother's anxiety, and may thus afford a certain degree of protection to the child. On the other hand analgesics and anaesthetics often prolong the duration of labour and may increase the need for operative delivery with consequent danger of birth injury and intranatal asphyxia. It should be recognised, therefore, that these drugs undoubtedly carry some risks to the baby as well as to the mother. This risk cannot be entirely eliminated even in the most skilled hands. When given in large dosage the drugs commonly employed may cause asphyxia and death of the child in utero or soon after birth; in some of these cases the drug is solely responsible while in other cases it serves to aggravate the harm caused by trauma, anaemia, pregnancy toxæmia or other factors. The premature baby is particularly vulnerable to the effect of analgesics and anaesthetics.

The possible ill effects of such drugs upon the child are:—

(i) *Asphyxia*

(a) *Before Birth.* The health of the foetus is dependent upon satisfactory oxygenation of the mother's blood. Many anaesthetics tend to cause some degree of maternal anoxaemia, and sometimes an anaesthetised patient becomes temporarily cyanosed because the air passages are obstructed. When this occurs the danger to the mother may be slight but the effect on the child may be serious or even fatal. Analgesics and anaesthetics also tend to depress the respiratory movements which the foetus makes in utero.

(b) *After Birth.* One of the risks to the baby is asphyxia neonatorum resulting from depression of the respiratory centre, and resuscitation of the infant is especially likely to be needed when certain types of analgesics are used, e.g., morphine-scopolamine and phenobarbitone. Fortunately, asphyxia neonatorum due to analgesics and anaesthetics is rarely fatal, though it may be alarming. When the drugs have been used in proper dosage response to treatment is usually prompt.

(ii) *Prolongation of Labour*

Nearly all the drugs employed for analgesic and anaesthetic purposes tend to inhibit uterine contractions and thus may prolong labour; on the other hand a careful choice of drug and proper timing in its administration may have the opposite effect. This is especially true in the first stage of labour when dilatation of the cervix may be hastened by removal of the inhibiting effect of the patient's fear of pain. In the second stage of labour, they often interfere with the patient's ability to co-operate by voluntary effort, and most of the reports on their use indicate an increased need for delivery by forceps or other operative measures. When labour is prolonged the risk of intrauterine asphyxia increases; when operative interference is required, the possibility of intracranial and other injuries arises.

(iii) *Cerebral Injury*

Schreiber, writing in 1938, expressed the view that foetal anoxaemia caused by large doses of anaesthetics given to the mother in labour may result in irreparable damage to the nerve cells of the brain, which in turn may give rise to various types of neurological disturbance later in life. However, Claye (1943), Kotz and Kaufman (1939) and other workers have been unable to confirm Schreiber's findings and it seems permissible to conclude that drugs used to relieve the pains of labour do not cause neurological disease in the child except in so far as they may influence the need for operative delivery. Nevertheless, bearing in mind the evidence (Chapter VI) suggesting that some forms of paralysis in infancy and childhood may be caused by asphyxia, it is well to remember the danger of overdosage of analgesics and anaesthetics.

There is no evidence to show that drugs administered to the mother during labour adversely affect the subsequent physical development of the child. The babies are sometimes drowsy during the first 24 hours, but their loss of weight during the early days is less than that of control infants (Kotz and Kaufman (1939) and Cole (1939)); and subsequently their gain in weight is at least as rapid as that of the controls. Breast feeding is not affected.

Conclusions

The best of the analgesics and anaesthetics, used with care and in the proper circumstances, do not ordinarily affect foetal or neonatal mortality and morbidity though they carry a slight risk. Their danger lies in their tendency to produce asphyxia and to prolong labour, thus increasing the need for instrumental delivery. Great care is necessary to avoid these dangers and the condition of the foetus and its oxygen requirements must be constantly in the mind of the anaesthetist. Although some forms of analgesia such as nitrous oxide and air with the percentage of each controlled by a special machine, have been shown to be so safe that they can be used by suitably trained midwives, the administration of anaesthetics in midwifery is a special subject demanding the services of an expert if it is to be done with safety to both mother and child. In our opinion the appointment of full time resident anaesthetists, with obstetrical experience, should be considered by all maternity hospitals. It is not proposed to discuss the relative merits of the various analgesics and anaesthetics in midwifery but it should be noted that the ideal method has still to be discovered. "The ultimate evaluation of any method of analgesia will depend on the price paid for the mothers' comfort in terms of infant mortality" (Sage, 1944).

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III. CONGENITAL MALFORMATIONS

INCIDENCE

It is difficult to estimate the incidence of congenital malformations and the part they play in causing neonatal death for the following reasons:— (1) Published statistics deal for the most part with hospital cases, i.e., with selected material, and do not necessarily indicate the incidence amongst the population in general. (2) Some reports deal only with cases in which malformations cause death; others include the less gross abnormalities which are compatible with life though not with health. (3) When a malformed child dies it is not always stated whether death was due to the malformation or whether the deformity was only an associated factor or a mere coincidence. Indeed, it is not always possible to decide this point even when a post-mortem examination is carried out.

Murphy (1940) found in Philadelphia that the frequency of malformed babies (born alive or dead) was approximately 4.7 per 1,000 live births, Malpas (1937) reported a general incidence of 21 per 1,000 amongst 13,964 consecutive births in the Liverpool Maternity Hospital during the years 1923-32 (material selected to some extent). The incidence in the same hospital during the years 1927 to 1942 was 31 per 1,000 (30,156 total births); but malformation caused death of the baby before or after birth in only 12 per 1,000 births.

Estimates of the incidence of congenital malformations amongst children born alive also vary. Murphy (1940) found it to be 5.4 per 1,000 amongst 122,654 live births in Philadelphia; Allen, Macafee and Biggart (1943) reported 43 fatal cases of foetal malformation amongst 6,314 babies born alive in Belfast (7.3 per 1,000). In the Liverpool Maternity Hospital, during the years 1927 to 1942, 635 of 28,153 infants born alive (23 per 1,000) showed some obvious malformation, but in only 84 (2.9 per 1,000) did death result from the deformity during the first two weeks of life. The Report (1945) of the Simpson Memorial Maternity Pavilion, Edinburgh, records 56 examples of congenital defect among 2,800 viable live births (20 per 1,000); 19 of these cases (7 per 1,000) died as a result of deformity. The Registrar General's Reviews of England and Wales for the years 1941 and 1942 show that for every 1,000 live births 4.05 and 4.09 babies respectively were registered as dying of congenital malformations within the first month of life (see Table). These figures must be accepted with caution, however, because it is likely that many were not verified by post mortem examination.

ENGLAND AND WALES

Year	Live Births	Neonatal Deaths	No. of neonatal deaths due to congenital malformation	Percentage of neonatal deaths due to congenital malformation	Incidence of fatal malformation per 1,000 live births
1941 ...	579,091	16,746	2,349	14.02	4.05
1942 ...	651,503	17,676	2,671	15.11	4.09
1945 ...	679,937	16,910	2,372	14.03	3.47

In every series of neonatal deaths it will be found that congenital malformations appear as an important cause. Dobbs (1943) reported that 11.0 per cent. of neonatal deaths were due to malformations and the records of the Liverpool

Maternity Hospital (1936—42 inclusive) show a similar figure (50 out of 359 neonatal deaths—11.1 per cent.). In a series of 339 neonatal deaths recorded by Baird (1945) developmental abnormalities accounted for 22 (8 per cent.). Cruickshank (1930) studied a series of 800 neonatal deaths and found 50 babies showing developmental abnormalities, but he considered that the deformity was sufficiently gross to have caused death in only 22 (2.75 per cent.). On the other hand Allen, Macafee and Biggart (1943) found that 43 of 211 neonatal deaths (20.4 per cent.) were due to malformations.

Macgregor (1946) studied 618 neonatal deaths and 453 stillbirths in Edinburgh (all submitted to autopsy), and concluded that the chief causes of death were:

Asphyxia	...	...	...	...	243 cases—22 per cent.
Birth Injury	...	...	...	...	276 cases—26 per cent.
Infection	...	...	...	...	204 cases—19 per cent.
Malformations	...	...	...	...	153 cases—14 per cent.
Other causes	...	...	...	...	195 cases—18 per cent.

Potter and Adair (1943) reported on 559 neonatal deaths and 614 stillbirths (autopsy in 81 per cent.) and found the chief causes of death to be:

Cause of Death	Antepartum Death	Intrapartum Death	Neonatal Death	Total
Anoxia, including placenta praevia, prolapsed cord, etc. ...	127 (34.2%)	110 (45.0%)	100 (18.0%)	337 (28.7%)
Birth Injury ...	—	33 (13.7%)	119 (21.3%)	152 (13.0%)
Major malformations ...	29 (7.8%)	36 (14.9%)	65 (11.3%)	130 (11.1%)

Developmental abnormalities were found to have caused death in 13.6 per cent. of the 954 cases submitted to autopsy.

Published statistics regarding congenital malformations vary to some extent according to the material subjected to analysis, and it should be noted that, since developmental faults cannot as yet be prevented, their incidence as compared with preventable causes of death (e.g., birth injury, prematurity), will vary directly with the standard of medical and nursing care, and with other factors such as the economic status of the mothers. According to Potter and Adair's classification (1943) congenital malformations have already taken third place in the list of the most important causes of neonatal death. Moreover in *two small series* of women in good circumstances and receiving the best obstetrical care, reported by Baird (1945), 40 per cent. of the stillbirths were due to malformation of the foetus. In the same city (Aberdeen) congenital malformations accounted for only 4.27 per cent. of stillbirths amongst women of equal social status but receiving less efficient medical and nursing attention.

It should be added that a considerable, though as yet undefined proportion of abortions is believed to be due to foetal malformations (Mall (1917), Huntington (1929), Schultze (1940), Corner (1944)). Indeed it has been suggested that more efficient treatment of threatened abortion may result in a reduced number of abortions but an increased number of viable malformed children.

If any generalisation is permissible it would seem to be that the incidence of fatal malformations amongst babies whose intrauterine development continues for 28 weeks or longer, is probably about 0.5 to 1 per cent., although

it is considerably higher than this in the practice of some hospitals. Of babies born alive approximately 5 per 1,000 die of malformation in the neonatal period; 10 to 20 per cent. of stillbirths and neonatal deaths are the result of developmental defects, but even higher figures may be found when all other factors are conducive to a lower general foetal mortality rate. Indeed, if the neonatal death rate be ever reduced to 10 per 1,000—a figure which seems feasible—malformations may become responsible for about half the neonatal deaths, unless in the meantime some method of preventing them is found.

CAUSATION

The aetiology of congenital abnormalities is a complex problem which has been reviewed recently by Corner (1944), Dunn and Salter (1944) and Morison (1945). Much remains to be determined but the evidence to date suggests that although the environment of the developing foetus may sometimes be a factor, hereditary influences are more important.

The part played by hereditary factors is seen most commonly in minor malformations such as hare lip and certain defects of the limbs which are known to recur in successive generations and which have been produced experimentally in animals by selective breeding. It is also generally realised that the offspring of any one couple may show a tendency to gross malformations such as anencephalus and spina bifida. Regarding the recurrence of monstrosities in the same family, Murphy (1936) found that in a series of 501 mothers who had each given birth to a congenitally malformed child over 50 per cent. did not conceive again; and of the remainder 12.4 per cent. gave birth to one or more additional malformed children, though not necessarily suffering from an identical lesion. The same author found that of 431 conceptions following the birth of a malformed child, 76.8 per cent. ended in the birth of a normal child at term, 10 per cent. in the birth of a congenitally deformed child, and the remainder in miscarriage, premature birth or stillbirth. Other clinical observations indicate that malformations are more common in white races than in coloured, and in mothers over the age of 30, especially those over the age of 40 (Malpas (1937), Penrose (1932), Potter and Adair (1943)). After a period of sterility there is an increased risk of a malformed infant being born (Murphy (1940)). Miscarriage, premature births and stillbirths are more often preceded or succeeded by the birth of a malformed child than would be expected normally.

Clinical observation and animal experiments reveal that many malformations are due to one of the sex cells carrying a lethal or detrimental gene. If this acts on one or other "organiser" at an early stage in development, gross malformations result; if it acts later, minor defects occur. Increased gene mutation rate following exposure of parental or grandparental gonads to radium or X-rays may be a factor in some cases.

As opposed to genetic or other inherent defects in the ovum or spermatozoon, various environmental factors acting on the early embryo (or in some cases possibly on the ovum prior to fertilization) have from time to time been suggested as causes of foetal malformation. For instance, maternal dietetic deficiencies and general ill-health have been blamed. With regard to the latter, White (1935) found that 7 out of 166 babies (4.2 per cent.) born of diabetic mothers were deformed. More recently has come the observation that rubella in the early weeks of pregnancy can result in the birth of children with developmental defects in the eye, ear and heart especially (Swan 1944, Parsons 1946, Clayton-Jones 1947). Exposure of the ovum or early embryo to X-rays or radium can also be a cause. Endocrine disturbance, leading to faulty embedding and nutrition of the developing ovum, remains a hypothetical

possibility. The effect of local environment and faulty implantation of the ovum is best seen in cases of tubal pregnancy when the foetus is frequently deformed (Mall 1908), and possibly in the condition of placenta praevia when again the incidence of malformation is believed to be increased. Greenhill (1939) found 2.5 per cent. monsters among 4,446 cases of placenta praevia, whereas there was only 0.94 per cent. among all cases. It should be added, however, that Potter and Adair (1943) found that among 95 foetal deaths in cases of placenta praevia, only 4 were due to malformations (about 4.0 per cent.), whereas among 1,078 cases of stillbirth and death unassociated with placenta praevia, the incidence of malformation was 11.8 per cent. Other environmental disturbances such as variations in the amount of liquor and the presence of amniotic adhesions are now considered the result rather than the cause of foetal malformations.

PREVENTION

So little is definitely known about the cause and mechanism of production of malformations that there seems little hope at present of reducing their incidence except by eugenic methods. The encouraging of child bearing at an early age and a general improvement in the health and nutrition of prospective parents might reduce the number to a limited extent, but further research is needed and would offer great scope.

CONCLUSIONS

Foetal malformations, though sometimes due to environmental factors, are most commonly caused by an inherent fault, either in the ovum or in the spermatozoon—a fault which is usually genetic. Advanced age or actual disease of the parents appears to play a part sometimes. It should be emphasized, however, that in all cases major deformities occur only if the adverse factors operate during the very early days or weeks of antenatal life.

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IV. MATERNAL HEALTH—SOCIAL AND ECONOMIC FACTORS

(a) AGE AND PARITY OF MOTHER

The stillbirth and neonatal mortality rates are both influenced by maternal age and parity. The stillbirth rate is relatively high in a first pregnancy, is at its lowest in the second and then slowly rises again until in the fifth it is as high as in the first; thereafter it rises still further with each succeeding pregnancy. The same general statement is true of the neonatal mortality rate, though the variations are not so marked. The total wastage by stillbirth and neonatal death is higher in multiparous women than in primigravidae. Thus Potter and Adair (1943), in an extensive investigation, found the combined stillbirth and neonatal mortality rate to be 3.4 per cent. for primigravidae as compared with 5.2 per cent. for multiparae. The neonatal mortality rates for these two groups were 1.72 per cent. and 2.43 per cent. respectively.

In each parity group the stillbirth rate, and to a somewhat lesser degree, the neonatal mortality rate, rises steadily with maternal age. Thus the stillbirth rate in primigravidae under the age of 25 is about half that for primigravidae of the 30-34 age group. This is shown by Baird's (1945) analysis of 8,808 children born in hospital in Aberdeen.

STILLBIRTH RATE PER 1,000 BIRTHS

Parity	Maternal Age			
	Under 25 years	25-34 years	Over 35 years	All ages
1st Pregnancy	20.2	53.4	83.3	33.6
2nd Pregnancy	12.8	20.3	59.2	20.0
3rd, 4th, 5th Pregnancy ...	13.7	32.1	33.2	29.1
6th, 7th, 8th Pregnancy ...	0.0	30.4	49.8	38.5
9th Pregnancy	—	105.3	67.6	73.5
All parities	17.7	35.8	53.5	30.4

NEONATAL MORTALITY PER 1,000 LIVE BIRTHS

Parity	Maternal Age			
	Under 25 years	25-34 years	Over 35 years	All ages
1st Pregnancy	32.7	45.3	39.0	37.0
2nd Pregnancy	29.5	26.1	48.9	29.0
3rd, 4th, 5th Pregnancy ...	33.4	28.6	40.1	31.4
6th, 7th, 8th Pregnancy ...	0.0	48.8	52.4	49.6
9th Pregnancy	—	58.8	36.3	40.7
All parities	32.0	34.6	43.4	34.5

Potter and Adair's (1943) analysis of a series of births in America also indicates the effect of maternal age.

Age of Mother	Distribution of Births	Distribution of Deaths (stillbirths and neonatal deaths)	Neonatal Mortality	Total Foetal Mortality
15-19 ...	7.8 per cent.	5.7 per cent.	1.8	3.2
20-24 ...	32.0 " "	23.7 " "	1.7	3.2
25-29 ...	29.6 " "	29.3 " "	1.9	4.3
30-34 ...	20.4 " "	22.1 " "	2.2	4.7
35-39 ...	8.3 " "	14.0 " "	3.6	7.4
40-44 ...	2.0 " "	5.3 " "	3.1	10.8

The effect of maternal age and parity is again shown in the Registrar-General's Statistical Review of England and Wales for 1938 and 1941. No figures are available for the corresponding neonatal death rates, but they would undoubtedly run parallel.

STILLBIRTHS AMONGST LEGITIMATE SINGLE BIRTHS

	Age under 25 (per cent.)		Age 25-34 (per cent.)		Age 35 and over (per cent.)			
	1938	1941	1938	1941	1938	1941		
1st Pregnancy ...	3.0	2.6	4.5	3.9	8.7	7.0		
2nd " ...	1.8	1.7	2.3	2.2	4.2	3.8		
3rd " ...	} 2.0	}	2.6	2.5	4.8	4.2		
4th " ...			2.7	2.6	5.0	4.3		
5th " ...			3.3	2.9	5.5	4.9		
6th " ...			4.1	3.3	5.4	5.0		
7th " ...			}	}	}	}		
8th " ...	4.1	3.8					5.5	5.8
9th " ...	}	}					}	}
10th " ...			4.5	5.7	5.7	6.4		
11th-15th ...			6.1	6.0	7.4	6.7		
16th and over ...					10.8	6.4		

The following table, kindly supplied by Dr. P. Stocks (Office of the Registrar-General), provides further evidence regarding stillbirths per 1,000 total births (lives and still) according to Birth Order and Maternal Age in 1939 and 1945 for England and Wales.

Order of Birth :—

Mother's Age	Year	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th and later	Total (Single births)	Total (including multiple births)
Under 20 ...	{ 1939 1945	27 19	18 11	33	—	—	—	—	—	—	—	—	26 18	27 19
20-24 ...	{ 1939 1945	30 22	18 13	23 16	28 19	27 27	30 40	45	—	—	—	—	26 19	27 20
25-29 ...	{ 1939 1945	38 27	21 14	25 17	29 21	32 24	34 40	25 23	70 67	53 75	22 83	18	30 21	31 22
30-34 ...	{ 1939 1945	55 40	28 19	31 22	37 25	37 27	35 25	41 33	38 40	56 49	71 28	40	39 27	40 27
35-39 ...	{ 1939 1945	80 54	37 28	40 32	48 34	48 37	49 68	52 40	53 41	57 40	76 50	64	51 36	52 37
40-44 ...	{ 1939 1945	113 74	52 35	60 45	56 55	67 53	68 45	66 59	63 57	67 68	74 55	73	67 52	68 52
45 and over	{ 1939 1945	144 110	92 40	103 72	73 83	83 73	84 40	96 72	96 34	121 100	90 83	92	95 71	97 73
All Ages ...	{ 1939 1945	40 29	24 18	31 24	38 30	42 34	45 37	50 43	54 47	63 54	76 51	70 55	36 26	37 27

The rates in bold type are based on less than 20 stillbirths.

In the first pregnancy the special risks to the baby are birth injury and maternal toxæmia (often with premature birth). The likelihood of difficult labour and toxæmia is reduced in the next few pregnancies, but thereafter birth injury again becomes an important cause of death, for the babies tend to be larger and to take up abnormal positions and the expulsive action of the uterus may become less efficient (Robinson (1930)). After the fourth or fifth child there is an increased risk of premature birth, and of antepartum hæmorrhage. As the size of the family increases other problems arise, depending mainly on whether the economic status of the mother keeps pace with the enlarging family. Many mothers of large families are over-worked, especially when they have no assistance in caring for their children; repeated pregnancies which are inadequately spaced add to the dangers of maternal malnutrition, exhaustion and chronic anaemia. The ill effects of general illness of the mother may be felt by the baby in utero and premature birth may result. Furthermore, the mother's ill health may impair her ability to breast-feed the child, and may prevent her giving it proper care and attention.

The effect of maternal age upon child bearing shows itself in many ways, but in general it can be stated that all the organs of the younger woman are more easily adapted to this function. Toxæmia of pregnancy is less likely in youth. The character of labour is more likely to be normal in the young mother. The uterus often becomes inefficient in its expulsive powers when middle age is reached and the baby of the elderly primigravida is especially prone to suffer birth injury.

The ideal family, from the standpoint of a low foetal mortality would be four or five children, with the mother not more than 24 when she has her first child, and not more than 35 when she has her last. Baird (1945) suggests that a woman's reproductive efficiency is beginning to wane even by the age of 20 in the poorer classes of the community. In the upper classes, however, efficiency may be maintained until the age of 30.

(b) ILLEGITIMACY

It has long been known that the stillbirth rate and the neonatal and infantile death rates amongst illegitimate babies are much higher than amongst those born in wedlock. This is borne out by the following statistics abstracted from the Registrar General's Statistical Review of England and Wales.

Year	Status	No. of Live Births	No. of Stillbirths	Stillbirth Rate per 1,000 births	No. of Neonatal Deaths	Neonatal death rate per 1,000 live births
1939	Legitimate ...	593,410	23,034	37·3	16,099	27·1
	Illegitimate	25,942	1,275	46·8	1,302	50·2
1940	Legitimate ...	581,156	21,427	35·5	16,375	28·2
	Illegitimate	25,873	1,304	48·0	1,128	43·6
1941	Legitimate ...	548,033	19,384	34·2	15,486	28·3
	Illegitimate	31,058	1,492	45·8	1,260	40·6
1942	Legitimate ...	615,036	20,835	32·8	16,200	26·3
	Illegitimate	36,467	1,548	40·8	1,476	40·5

According to the above figures approximately one in 24 children born in the years prior to the War was illegitimate. The relative frequency is now likely to be higher* and illegitimacy must be responsible for the deaths

* For example, in Liverpool before the recent War the illegitimate rate was 4·8 per cent. of births, whereas during 1946 the rate had increased to approximately 8·0 per cent. In Birmingham approximately 10 per cent. babies born during 1946 were illegitimate and in one third of these cases the mother was already married.

of several hundred babies each year. The reasons are clear: lack of care of the mother during pregnancy and labour, and lack of care of the newborn baby by the mother. The neglect of the mother is usually the fault of the woman herself and of her relatives, who tend to prefer secrecy to medical and nursing attention; both mother and baby suffer on this account. The baby is usually unwanted; the mother is less inclined to breast-feed it, and may be prevented from doing so by the necessity for earning a living. Often it is in the care of those who have not sufficient interest in its welfare to give it the best chance of survival.

The solution of this problem is largely one of education—education of the young in matters of sex and in the proper employment of leisure. Facilities for healthy recreation must be provided as well as proper living conditions at home. There should be efficient medical and nursing care available for all unmarried mothers and their babies; and the general care of these women should be directed by those who can show the necessary sympathy and encouragement. When the baby is unwanted and there seems little chance of its being properly cared for, adoption should be arranged as early in its life as possible.

(c) ECONOMIC CONDITIONS

There is clear evidence that economic and social factors play an important part in both stillbirth and neonatal mortality. The Registrar-General recognises five major social classes made up as follows:—Class I, members of the professions, commissioned officers and well-to-do people concerned with shipping, finance, etc.; Class II, intermediate between Class I and skilled workers; Class III, skilled workers; Class IV, intermediate between skilled and unskilled workers; Class V, unskilled workers. In the period 1930-32 the neonatal death rate among legitimate children in England and Wales was 50 per cent. higher in Class V than in Class I, and the stillbirth rate was about 30 per cent. higher, while the infant mortality rate for the age group 6-12 months was four times as high. The figures for the neonatal death rate for the 1930-32 period are:

All Classes	Class I	Class II	Class III	Class IV	Class V
30.2	21.7	27.2	29.4	31.9	32.5

In so far as the total loss of child life is concerned, these figures become even more significant when it is realised that Class V is a much larger group than Class I, and in any case the gross reproduction rates in the two classes at that time were about 1.3 and 0.7 respectively. The results of a special analysis for 1939, kindly supplied by the Registrar General for England and Wales, are set out in the following table and confirm the findings of the earlier period.

England and Wales, 1939	Class I	Class II	Class III	Class IV	Class V	All Classes
Stillbirths per 1,000 live births:						
(a) Crude	24.4	33.4	35.6	37.6	39.7	36.2
(b) With fixed weighting by mother's age and birth order	24.0	31.7	35.8	38.8	40.3	36.2
Neonatal Mortality	18.9	23.4	25.4	27.7	30.1	27.1

The stillbirth and neonatal mortality rates also vary according to the different localities and regions, viz.:

	Stillbirths per 1,000 total births			Neonatal Death rate			Infant Mortality rate		
	1938	1941	1942	1938	1941	1942	1938	1941	1942
England and Wales ...	38	35	33	28	29	27	53	60	51
Greater London ...	31	30	29	23	27	25	50	52	46
Rest S.E. England ...	34	29	28	24	24	23	42	44	40
East ...	37	34	32	27	25	26	44	47	45
Midlands ...	38	34	33	29	29	27	52	60	49
South West ...	39	32	32	28	26	25	47	51	42
North ...	42	39	47	32	32	30	60	72	60
Wales ...	51	43	40	33	33	31	57	70	58
Scotland ...	42	40	38	35	38	34	70	83	69
Northern Ireland ...				35	34	35	75	77	76

This table shows that in some areas, particularly the North, Wales and Scotland, both stillbirth and neonatal mortality rates are considerably higher than in London and the South. Moreover, if areas where the stillbirth rate is low are compared with areas where it is high, it is found that the mortality in each age group runs parallel with the total. For example, in the age-group 20-24, the stillbirth rate in 1938 in Greater London and New York State was 22 and 21 respectively, whereas for Wales and North of England it was 35 and 35. It is clear, therefore, that in Wales and the industrial areas of Britain such as the North of England, Glasgow and Clydeside, the stillbirth, neonatal and infantile mortality rates are high. In these places factors making for high foetal death rates must operate even in favourable age groups, and amongst these influences may be mentioned unemployment (prior to the War), poverty, bad housing, malnutrition, ignorance, high fertility together with insufficient help in the home, and lack of preparation for marriage as shown by the high illegitimacy rate and high prenuptial conception rate. In 27 of the large cities of Britain in 1936 the stillbirth rate was 45 per 1,000 when 6 or more per cent. of the population were unemployed, and 37 per 1,000 when there were 5 per cent. or less unemployed. The corresponding figures for neonatal mortality were 35 and 28. Rietz (1930) in a study of legitimate births in Stockholm found the stillbirth rate was 14.3 when the father's income was £600 p.a. or more, but rose to 48.9 when the income was less than £240 p.a. The neonatal death rates in these two groups was 11.4 and 24.0 respectively. Baird (1945) has shown that the main reason for the high neonatal death rate in Scotland, as compared with Holland, and for Social Class V of England and Wales as compared with Social Class I, is the relatively high proportion of premature births; this in turn is associated with poor social and economic conditions affecting adversely the physique, health and nutrition of the mother.

Direct comparison of death rates in the different social classes and in various geographical areas is not easy because several factors are concerned. Thus it is found that there is only about a 30 per cent. difference between the stillbirth rates in Classes I and V, but the causes of death are different. In Class I there is a lower death rate from prematurity, "congenital debility" and antepartum haemorrhage, and a higher death rate from birth injury. The neonatal death rate due to malformations is roughly the same in two groups.

It has been found that there is relatively little pre-eclampsia and premature labour in private as opposed to hospital practice, and comparatively few cases of contracted pelvis occur; furthermore there is little anaemia or malnutrition. These findings compensate for the disadvantage of a high proportion of elderly primigravidae in the higher income classes. It must be noted, however, that except where the standard of obstetric skill is high, the intranatal and neonatal death rate in this class is relatively high, mainly owing to birth trauma. Other factors which affect the comparison of statistics are the standard of medical and nursing care and hospital facilities which are available and utilised in different regions and for different grades of society. These factors have been excluded by Baird (1945) in a survey of two comparatively small groups of patients (1) Private, (2) Hospital—both under the care of the same expert obstetricians.

Maturity	Stillbirths	Stillbirth rate per 1,000 births	Neonatal deaths	Neonatal death rate per 1,000 live births
Hospital Class, 8,806 cases :				
Full-time, 7,999, 90.8% ...	132	16.5	131	16.7
Premature, 809, 9.2% ...	136	168.1	364	243.7
		30.4		34.5
Private Patients, 501 cases :				
Full-time, 481, 96.0% ...	4	8.3	3	6.3
Premature, 20, 4.0% ...	1	50.0	1	52.6
		10.1		8.1

The only difference between these two groups was economic status. In both, difficult labour was the chief cause of death, and was due to contracted pelvis in the hospital class, while in the private series it appeared to be related to the high frequency of elderly primiparity. The difference in the results in the two groups is explained for the most part by the high incidence of prematurity amongst the women of lower social grade.

Amongst the well-to-do, despite the disadvantage of a relatively greater number of primigravidae and increased age at maternity, it is possible by first class medical and nursing care to attain low stillbirth and neonatal death rates. Thus Baird (1945) in Aberdeen reports two series with neonatal death rates of only 8.1 and 13.7 per 1,000. Allen, Macafee and Biggart (1943) in Belfast, and Rietz (1930) in Stockholm record rates of 9.4 and 11.4 respectively.

(d) DIET IN PREGNANCY

Stockbreeders and veterinarians have long been aware of the importance of a good diet during pregnancy and lactation in animals. Dairy farmers have shown that milk production varies with the nature of the diet, particularly in the later part of pregnancy. There is ample evidence that fertility, dentition, the form of the bony pelvis, the size and well being of the foetus in utero, the process of labour itself and the susceptibility of the animal to infection, are, to a considerable extent, dependent on the food with which it is fed. This knowledge applies equally well in the human field.

Pregnancy is a "diet efficiency" test and during this period of physiological strain, border line states of malnutrition may be revealed which otherwise would have remained latent. During the last three months of pregnancy 70 per cent. of the birth weight of the mature foetus is deposited. During these months it receives from the mother 75 per cent. of its protein, 93 per cent. of its fat, 65 per cent. of its calcium, 68 per cent. of its phosphorus, and 80

per cent. of its iron content. (Figures supplied by St. George Huggett, of the Department of Physiology, St. Mary's Hospital, London.) The foetus makes a particular demand upon the mother's iron and calcium, and it is dependent on the mother's food supply for essential amino-acids and vitamins, especially the fat-soluble vitamins.

One of the most recent observations on the effect of maternal malnutrition upon the foetus is referred to by Barcroft (1946) and was made by Clement A. Smith, who carried out a survey in Holland during 1945-1946. He concluded that it is in the last trimester of human pregnancy that diet influences the weight of the foetus most definitely. In a community where diet is faulty or restricted, the children and mothers, expectant and nursing, suffer first. This does not mean, however, that the baby born of an under-nourished woman is necessarily small and feeble, and it is a common experience in obstetrics to see ill fed and unfit women give birth to babies who are apparently healthy and of average weight. Reid and Mackintosh (1937) found that poor social conditions did not influence the birth weight of babies. The child in utero may behave as a parasite, obtaining its nourishment from the breakdown of the host tissues if need be. Yet there is a considerable mass of evidence showing that the nutritional standard of expectant mothers has a bearing upon the health of their babies. In this connection there have been four large scale clinical investigations in recent years.

(I) Since 1934, the National Birthday Trust Fund has sponsored "field" investigations in some of the depressed areas of England and Wales. Supplements of milk and yeast were given to one group of expectant mothers while another comparable group received minerals and vitamin concentrates. In the group which took the milk and yeast there was a significant reduction in the number of stillbirths and neonatal deaths. (Balfour 1944.)

(II) In a Toronto experiment (Ebbs and others, 1942), expectant mothers were given daily supplements of proteins, fats, fruit, vitamin concentrates and iron. When compared with a control group on a relatively poor diet the women who received supplementary foods had fewer miscarriages and premature births, and were less liable to toxæmia of pregnancy; and their babies were less subject to colds, pneumonia, rickets and anaemia. Breast feeding both in hospital and at home was possible in a greater proportion of those women who received additions to diet.

(III) The People's League of Health (1946) made a study of 5,000 expectant mothers attending 10 London hospitals between March 1938 and the end of 1939; an initial survey of 1,000 of these women showed that the diet was deficient in calcium in 70 per cent. and deficient in iron in 90 per cent. One half of the whole group received supplements of calcium, iron, iodine and vitamins A, B, C and D, from before the 24th week of pregnancy to the time of delivery; the other half was the control group. The most significant finding was a reduced incidence of prematurity and of toxæmia of pregnancy in the treated group; thus, among 1,529 primigravidae receiving additional diet the pregnancy ended prematurely in 308 (20.1 per cent. $\pm$ 1.10), while prematurity occurred in 361 (23.9 per cent. $\pm$ 1.10) of 1,512 primigravidae whose diet was not modified. The corresponding figures for the multiparae were of a similar order—20.1 $\pm$ 1.33 and 24.2 $\pm$ 1.33. The differences in both instances are greater than could be accounted for by chance fluctuations. The primigravidae who received the additional diet had an incidence of toxæmia of 5.4 per cent., while in the primigravidae whose diet was not augmented the incidence was 7.4 per cent. The difference and its standard error was 2.0 $\pm$ 0.9, which means that the odds are nearly 40 to 1 against such a result being obtained by chance. The results for parous women

were also favourable, though not to a significant degree, the percentage being 3.6 and 5.2 respectively, the difference being 1.6 ± 0.9 . These results suggest that the danger of toxæmia to women receiving the special diet was reduced by 30 per cent. The investigation was carried out in an area in which the incidence of toxæmia is known to be low, and a reduction of 1.8 per cent. was obtained. It is estimated that if a similar reduction could be obtained for the whole country the number of cases of toxæmia in England and Wales in a year would be reduced by 10,000. The effect of toxæmia upon the stillbirth rate and the neonatal death rate is discussed later in this report.

(IV) Toverud (1939) has described an investigation which was carried out at a health centre in a rural community (Skodsmo) in Norway. The health of pregnant women and their children was supervised and it was found that about one-third of the women were anaemic and their diets were deficient in calcium and vitamins A and C. With proper feeding these deficiencies were corrected before delivery. It was found that the incidence of eclampsia and stillbirths was reduced in those women who received dietetic supplements and the incidence of premature births was reduced from 8 per cent. to 2 per cent. In a group of unmarried mothers who were given an adequate diet the number of pregnancies which ended prematurely was reduced from 15 per cent. to 2 per cent.

Lactation was also influenced beneficially by proper feeding during the pre- and post-natal periods and even among the unmarried women breast feeding was possible in 90 per cent. The children of those women who attended the health centre showed a marked diminution in the incidence of rickets. When the children were between two and three years of age dental caries was present in 30 per cent. as compared with 82 per cent. in the group whose mothers had not attended the health centre.

The results of these investigations which demonstrate the beneficial effects of a well regulated diet upon the health of the mothers and the development and well being of their infants are of national importance. Under-nourishment may be due to defects of quality as well as of quantity of diet, and malnutrition occurs not only in the lower income classes, but sometimes amongst members of more favoured classes when diet is unbalanced. Nevertheless, poverty and malnutrition usually march hand in hand. For the expectant mother the quality of the diet is of special importance and it has been shown that the intake of calcium, for example, by many pregnant women is less than half their requirement. The necessity for extra proteins, minerals and vitamins in the diet of expectant mothers and children has been recognised by the Government during the war, and the availability of these valuable foods is now a question of needs rather than of ability to pay. The fall in stillbirth and infant mortality rates which occurred generally throughout the country during the war years may be due, at least in part, to the greater care taken of the health and nutrition of pregnant women and of their babies. Increased purchasing power must also have had a beneficial effect.

There is good reason to believe that a greater improvement in social and economic conditions, with raising of nutritional standards, would result in a further reduction in the stillbirth and neonatal death rates. The subject is one which must be viewed broadly if the best results are to be obtained. Special care of a woman's health and nutrition should not be deferred until she becomes pregnant—it should be continuous throughout the whole of her life. Contracted pelvis, toxæmia and other conditions which threaten the life and health of the next generation will largely disappear when every child has healthy parents, is born and reared under good conditions and receives a proper education.

(e) MATERNAL DISEASES

(i) *Nutritional anaemia of pregnancy:*

The frequency of iron deficiency anaemia during pregnancy varies in different parts of the country. For example, it was shown before the recent war that 17.5 per cent. of a group of pregnant women in Aberdeen had a haemoglobin level below 70 per cent. whereas the figures for London and Stockport were 4.6 per cent. and 10.2 per cent. respectively.

In 1937 Reid and Mackintosh attempted to record the influence of anaemia and poor social circumstances during pregnancy on the subsequent history of mother and child in 1,094 women attending the Stockport ante-natal clinics. They concluded that although anaemia and poor social circumstances did not appear to affect the birth weight of the babies, they had some slight influence on the stillbirth rate among the primigravidae and a definite ill-effect upon the neonatal and infantile death rates. Neither anaemia nor social circumstances appeared to have much influence on the establishment of breast feeding. When, however, breast feeding was actually established, women in a low social group (average net income, after deduction of rent, 12s. per head per week or less) with a haemoglobin reading of under 70 per cent. were unable to maintain breast feeding for as long as women in better social circumstances and with a more satisfactory haemoglobin level. Among the poorer mothers, severe anaemia during pregnancy was associated with an increased sickness rate among their children.

In a discussion on the war-time problem of nutritional anaemia in babies, Mackay and others (1942) brought forward evidence that anaemia in the baby is in part the result of anaemia in the mother during pregnancy. They referred to an investigation in America which showed that the babies of a group of mothers with severe anaemia during pregnancy (average haemoglobin 39 per cent.) had an average haemoglobin percentage of 50 at the age of one year. Other severely anaemic mothers were treated during pregnancy and their mean haemoglobin level was raised from 40 to 72 per cent. before term. Their babies, at 12 months of age, had a mean haemoglobin level of 77 per cent., a figure that is very similar to that of infants whose mothers' haemoglobin was considered normal. Mackay (1933) also showed that in a district in the East End of London the subnormal haemoglobin level of the infants was in part at least the result of nutritional anaemia in their mothers. She considers that if the haemoglobin level of pregnant women is lower to-day than it was before the war, there is likely to be a greater tendency to nutritional anaemia in their offspring.

Nutritional anaemia is a common cause of poor health in women and children and it results in lack of mental and physical vigour. Its cause is to be found not only in dietetic and hygienic deficiencies but in too-frequent pregnancies. Its prevention depends on proper feeding, better hygiene and adequate spacing of pregnancies.

(ii) *Toxaemia of pregnancy:*

The toxaemias of late pregnancy are the most important of the maternal diseases which affect antenatal, natal and neonatal mortality. The group includes eclampsia, pre-eclampsia, chronic nephritis, chronic hypertension and "recurrent toxaemia." Some clinicians would also include accidental ante-partum haemorrhage, but for simplicity it is excluded from the statistics quoted here. It is not easy to determine the part that toxaemia of pregnancy plays in neonatal mortality and morbidity, because most of the published

statistics deal only with the total foetal death rate, often including abortions; they rarely specify the number of neonatal deaths. Moreover, post mortem examination of the babies born to toxæmic women does not reveal any lesion which, in so far as is known at present, can be attributed directly to the toxæmia. For this reason many of the reports based upon findings at autopsy omit toxæmia as a cause of death (e.g. Potter and Adair, 1940). On the other hand, reports based upon clinical observations give to toxæmia an important place in the causes of foetal death. Thus Dunham and others (1938) investigating a series of 6,750 stillbirths gave toxæmia as a cause in 1,433 and as an associated factor in another 168. Such statistics suffer from the disadvantage that although toxæmia may have appeared to be responsible for death, autopsy might have revealed other causes.

Incidence of toxæmia in obstetric practice

The incidence of toxæmia in hospital practice varies from 3.5 per cent. to 10 per cent. of pregnancies in this country; it is rarely below 5.0 per cent. In Manchester it is 9.0 per cent., in Newcastle 8.2 per cent., in London 5.8 per cent. and in Liverpool 6.8 per cent. Over the country as a whole, i.e. including domiciliary practice, the incidence may be somewhat lower.

Total foetal mortality (abortions, stillbirths and neonatal deaths) associated with toxæmia of pregnancy.

The foetal mortality is illustrated by the following table which concerns toxæmias of late pregnancy (excluding chronic hypertension).

Source	Total No. of deliveries in period under review	Foetal mortality in toxæmic cases	Total foetal mortality for hospital (including toxæmia)
Manchester* ...	13,148	29.2 per cent.	12.4 per cent.
Newcastle* ...	8,414	46.9 " "	13.6 " "
London* ...	12,304	25.6 " "	8.2 " "
Liverpool* ...	14,028	21.5 " "	8.6 " "

* See note on p. 29.

In America Wellen (1940) reported a foetal mortality rate of 30.8 per cent. in 7,887 cases, and Scott (1940) a mortality rate of 27.2 per cent. Eclampsia is the most dangerous type of toxæmia from the standpoint of the child; American statistics which exclude this disease show a lower foetal loss (e.g. Peckham (1933) 14.9 per cent., Stander (1929) 15.8 per cent., Dieckman and Brown (1939) 12.8 per cent.); but American and British figures are not easily comparable because of different classifications of toxæmia. For this country the total loss of child life in eclampsia is between 40 per cent. and 60 per cent.; in albuminuria without convulsions (pre-eclampsia, nephritic toxæmia, recurrent toxæmia) between 20 per cent. and 30 per cent.; for all toxæmias, between 25 and 40 per cent. The Newcastle figures are unusually high and perhaps this is because the hospital in question deals with an unusually high proportion of emergency, as distinct from "booked" cases; but the depression and poverty in the Newcastle area at the time of the report may also have been a factor.

Neonatal mortality in toxæmia of pregnancy

Even though the major part of the foetal mortality is accounted for by abortions, antenatal and intranatal deaths, the death rate among infants

born of toxæmic mothers is two to four times as high as the neonatal mortality rate as a whole. Thus:—

Source	No. of cases of toxæmia	Abortions and stillbirths	Neonatal deaths	Neonatal death rate per 100 live births in toxæmia	Neonatal death rate per 100 live births for hospital at time of review
Manchester* ...	1,191	258	91	9.7	3.86
Newcastle* ...	533	79	71	19.8	4.5
London* ...	710	133	48	8.3	2.28
Liverpool* ...	954	167	39	4.7	2.63

* See note on p. 29.

Again from an analysis of all the stillbirths and neonatal deaths occurring in the hospitals under review, it appears that nearly 20 per cent. of the babies dying in the neonatal period are born to mothers suffering from toxæmia, as is shown in the following table:—

Source	Stillbirths, total number	No. of stillbirths associated with toxæmia	Total No. of neonatal deaths	No. of neonatal deaths associated with toxæmia
Manchester* ...	1,175	220 (18.8%)	460	91 (19.8%)
Newcastle* ...	806	166 (20.6%)	347	71 (20.5%)
London* ...	742	123 (14.0%)	264	48 (18.2%)
Liverpool* ...	851	167 (19.6%)	359	39 (10.9%)
Belfast* ...	598	216 (36.0%)	?	?

* See note on p. 29.

In assessing the figures in this table reference should be made to the incidence of toxæmia in the hospitals in question (see p. 27).

Factors concerned in foetal and neonatal death in toxæmia of pregnancy

(i) *Prematurity.* It has been noted that lesions attributable directly to maternal toxæmia cannot be found at autopsy upon the child dying either before or after birth. But the placenta is often small—especially in cases of chronic vasculo-renal disease—and its blood vessels and circulation are believed to be damaged (Falkiner and Apthorp, 1944). Sometimes there is placental separation with a retroplacental haemorrhage. It is assumed, though there is no proof, that such conditions frequently impair the nutrition of the foetus sufficiently to cause its death in utero. At other times the foetus is born alive, though prematurely; or, if born at term, it is often small and immature. Finally, and most important, the obstetrician often induces labour prematurely in the interests of the mother, and thus unavoidably exposes the child's life to risk; though it should be added that induction of premature labour is often in the best interests of the child and may prevent its death in utero.

The relationship of toxæmia of pregnancy to prematurity is illustrated by the following statistics from the literature, and by the figures quoted in chapter 8 (page 48). The figures show wide differences because the severity, methods of treatment, and the classification of toxæmia vary in different centres.

AMERICA	
Hess <i>et al</i> (1934)	... 16 per cent. of premature babies are born to toxæmic mothers.
Clifford (1934)	... 30 " " " " " " "
Massey (1940)	... 25 " " " " " " "

BRITISH ISLES	
Belfast* ...	... 49 per cent. of premature babies are born to toxæmic mothers (251 out of 513).
London*	... 30 per cent. of premature babies are born to toxæmic mothers (203 out of 663).
Liverpool*	... 21 per cent. of premature babies are born to toxæmic mothers (143 out of 673).
Dublin* ...	... 16.6 per cent. of premature babies are born to toxæmic mothers (40 out of 241).

* The figures quoted for hospital practice have been abstracted from the published reports of the following hospitals: Belfast, Royal Maternity Hospital (1932-1938); Liverpool, Liverpool Maternity Hospital (1936-1942); London, Queen Charlotte's Maternity Hospital (1932-1936); Manchester, St. Mary's Hospital (1934-1941); Newcastle, Princess Mary Maternity Hospital (1934-1937). In some respects the figures are approximate and not accurate because an allowance has had to be made for the fact that some cases of toxæmia were complicated by multiple pregnancy and the number of babies born was greater than the number of mothers. In the case of twins, the two children have as far as possible been regarded as one, each individual being entered as half a still-birth, neonatal death or live birth. The figures for Dublin are based on observations made in the Rotunda Hospital by N. M. Falkiner and communicated personally by him to the sub-committee. Unless otherwise stated, cases of hypertension without albuminuria are not included in the statistics.

The importance of prematurity or immaturity as a cause of death among babies born alive to toxæmic mothers is brought out by further details from the Liverpool reports. In 954 cases of toxæmia, 39 of the babies born alive died in the neonatal period. The birth weights of these babies were as follows:—

Weight*	1-2 lbs. (455- 909 gms.)	2-3 lbs. (909- 1364 gms.)	3-4 lbs. (1364- 1819 gms.)	4-5 lbs. (1819- 2274 gms.)	5-6 lbs. (2274- 2729 gms.)
Number of babies	2	12	10	8	7

None of the babies was over 6 lb. in weight, indeed only one was above the "immature" border-line of 5½ lbs. (2,500 gms.) hence 38 out of 39 were immature or premature.

* When a baby's weight was exact in pounds it was included in the higher group, i.e. a 2 lb. baby is put in the 2-3 lb. column.

To show that the foetal mortality in toxæmia is largely dependent upon the size of the baby, Dieckman and Brown (1939) give the following figures which include stillbirths as well as neonatal deaths:—

Weight of baby	Mortality in cases of toxæmia (per cent.)	Mortality for all cases in hospital (per cent.)
1- 999 grammes (less than 2.2 lbs.)	96	100
1000-1499 „ (2.2 to 3.3 lbs.)	71	73
1500-1999 „ (3.3 to 4.4 lbs.)	28	22
2000-2999 „ (4.4 to 6.6 lbs.)	8	
3000-3999 „ (6.6 to 8.8 lbs.)	3	
Over 4000 grammes (over 8 lbs.) ...		

It is permissible to conclude, therefore, that the most important causes of the high neonatal death rate of babies born to toxæmic mothers are prematurity and immaturity. Such babies are especially prone to suffer birth injury, particularly intracranial hæmorrhage, and intrauterine asphyxia as a result of defective placental function probably increases their susceptibility to intracranial injury.

Dieckman and Brown (1939) give figures showing that the mortality rate among viable prematures born to toxæmic mothers is 26 per cent. as compared with a mortality rate of 21.9 per cent. among viable prematures for all cases. The difference is in part explained by the high incidence of intracranial injury. Dieckman and Brown conclude that if the foetus is born alive and survives the first few days, maternal toxæmia will have no further influence upon its life and development. This is the general opinion, which receives further support in the observations of Hess and colleagues (1934); these writers concluded that if those babies having intracranial hæmorrhage were omitted, the development of the premature baby of the toxæmic mother appeared to be similar to that of other prematures.

(ii) *Severity of toxæmia.* Another factor influencing foetal and neonatal mortality in cases of toxæmia deserves mention, namely the severity of the toxæmia. Peckham (1933) showed that the mortality rate varied directly with the mother's blood pressure:—

Maternal Blood Pressure	Foetal Death Rate (per cent.)
Less than 149 systolic	9.0
150-179 systolic	11.0
180-209 systolic	19.0
Over 210 systolic	42.0

Clifford (1934) found the foetal mortality rate to be 50 per cent. when the maternal blood pressure exceeded 180 mm. Hg. Peckham (1933) also gives figures showing the relationship of foetal mortality to the degree of maternal albuminuria; and Browne and Dodds (1940, 1942) suggest that the foetal mortality varies with the severity of the disease in cases of chronic hypertension. The more severe the toxæmia, the greater the likelihood of premature labour—either spontaneous, or induced by the obstetrician.

(iii) *Mode of delivery.*—There is statistical evidence (Dieckman and Brown, 1934) demonstrating that the mode of delivery plays a part in determining the rate of foetal mortality; the small size of the foetus, its liability to anoxæmia both before and during delivery and its vulnerability to birth-injury all combine to increase the risks of operative interference such as version. Even slight delay of the head on a resistant perineum may be fatal. Episiotomy may save the child's life. Although the application of

forceps may in some cases cause intracranial injury, it may at other times be protective in that the risks of intranatal asphyxia developing are reduced. In recent years delivery of the child by Caesarean section in cases of severe toxæmia has been viewed more favourably by some obstetricians. Considering only the interests of the baby, there is some justification for stating that the smaller the baby, the greater the indication for Caesarean section. If this is done, however, the dangers of most types of anaesthesia to the immature child must be kept in mind and some form of local or regional analgesia probably offers the baby the best chance of survival.

Summary relating to toxæmia of pregnancy

The high neonatal death rate in toxæmia of pregnancy is primarily dependent upon prematurity or immaturity. The baby born to a toxæmic mother is exposed to all the risks of prematurity and is especially liable to intracranial haemorrhage, a tendency aggravated by antenatal and intranatal asphyxia.

The risk to the baby is dependent to a large extent upon the severity of the toxæmia and is at its highest in eclampsia. The mode of delivery is another important factor affecting prognosis.

The underlying cause of toxæmia is unknown and its treatment is largely empirical. Nevertheless there is evidence to show that a proper diet and healthy living conditions may lessen its incidence. Apart from attention to such matters, the chief hope of reducing the loss of child life is the early detection of the disease by adequate ante-natal care and by efficient treatment—treatment that takes cognizance of the foetus as well as the mother. Special care is called for in the technique of delivery.

(III) Other Maternal Diseases

Diseases affecting the mother may have harmful effects upon her child (i) by interfering with her ability to supply the needs of the developing foetus; (ii) by precipitating abortion or premature labour; or (iii) by interfering with the natural course of labour and the mother's ability to breast feed and to give adequate care to her baby. Each disease, e.g. cardiac disease (mainly of rheumatic origin); chronic bronchitis; asthma and other diseases of the lungs; pyelitis etc., presents its own special problems. Perhaps diabetes mellitus deserves special mention because the associated foetal mortality is exceptionally high. Thus Kramer (1935), who analysed 238 cases collected from the literature, found the stillbirth rate to be 25 per cent.; and Bill (1944) reported 7 stillbirths and 5 neonatal deaths in a series of 44 diabetic pregnancies—a total foetal and neonatal mortality rate of 27 per cent. Formerly the total mortality rate was approximately 50 per cent.; and in spite of special obstetrical care and treatment with insulin, the figure still stands at about 30 per cent. (see also Miller, 1945, 1946), although a few workers have reduced foetal mortality to approximately 15 per cent. (White and Hunt (1943); Hurwitz and Irving (1937); Mengert and Laughlin (1939).)

Syphilis receives special attention in Chapter IX.

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V. BIRTH INJURIES

Birth injuries may cause intranatal death, neonatal death and neonatal illness; they may also cause permanent mental or physical crippling in those infants who survive the initial damage.

(a) INCIDENCE OF BIRTH INJURIES

Cruickshank (1930) made post mortem examinations of 800 infants up to four weeks of age, and found that in 540 (67 per cent.) death was ascribable to asphyxia neonatorum, prematurity or birth injury. In most of the cases these conditions were combined; 400 of the infants were premature. When asphyxia and head injury occur together it is scarcely possible to state which is the primary and which the secondary lethal factor; head injury may lead to asphyxia, and asphyxia favours haemorrhage. The following figures illustrate the frequency with which intracranial haemorrhage has been found in stillbirths (macerated foetuses excluded) and neonatal deaths: Holland (1920) 167 stillbirths, 55 per cent.: Browne (1921) 92 stillbirths, 24 per cent.: 80 neonatal deaths, 44 per cent.: Capon (1922) 52 stillbirths, 46 per cent.: 28 neonatal deaths, 21 per cent.: Cruickshank (1923) 400 premature and mature stillbirths and neonatal deaths, 45 per cent.: Bundesen et al (1938) 1,043 neonatal deaths, 22.7 per cent.: McNeil (1940) 108 neonatal deaths, 37 per cent.: Evans and Stewart Smith (1946) 150 stillbirths and 223 neonatal deaths, 17.2 per cent.

Most observers agree that one of the leading causes of death in the first four weeks of life is birth injury, and that the majority of such deaths occur in the first week. That birth injury is the immediate cause of death in approximately one quarter to one third of all neonatal casualties would probably be accepted by most authorities; and it accounts for approximately one quarter of all cases of intranatal death. Wohlwill's warning is not an overstatement: "The loss of human life due to birth injury approaches in its social bearing the loss caused by any of the known and more common diseases affecting large masses, such as tuberculosis." Indeed, it can be calculated that in England and Wales the number of babies killed by injuries received during birth (stillbirths and neonatal deaths) in any one year is approximately twice the total number of deaths, adults and children, in road accidents.

(b) CAUSATION OF BIRTH INJURIES

When compared with the lower animals the human foetus is peculiarly liable to injury at birth. Even at the term of pregnancy it is relatively delicate and none of its tissues—bones, muscles, blood vessels or viscera—is capable of withstanding the application of much force. Furthermore, the birth canal of a woman is not straight like like of quadrupeds and consequently the foetus is exposed to prolonged and often dangerous stresses. Thus injuries are liable to occur even when obstetrical conditions are favourable. As might be expected, mechanical factors responsible for intracranial lesions occur more often in primigravidae than in multiparae. In addition there are other factors, increasingly common in recent times, that may act prejudicially: for example, elderly primiparity, the demand for shorter and more comfortable labours, the over-use of analgesic, anaesthetic and oxytocic drugs, the abuse of labour induction and of obstetric forceps.

The causation of birth injury is, in general, mechanical and its study demands consideration of the stresses of labour and the varieties of strain they may produce on the foetus. Aggravation of the immediate (mechanical) cause by certain conditions which may be described as *contributory* factors is not uncommon; obstetrical manipulations, methods of resuscitation and

the haemorrhagic diatheses may be instanced. In the post mortem studies by Potter and Adair (1939 and 1940) intracranial haemorrhage was found in the following percentages in different types of birth: natural cephalic delivery 6.4: Caesarean section 10.8: version and extraction 12.5: low forceps 24.5: mid and high forceps 69.1. The fact that fatal injuries can occur during a normal and easy delivery and even when the child is delivered by Caesarean section is not always appreciated. In Caesarean section cases, the injury may occur before the operation is done.

In addition to the immediate mechanical factor, two important predisposing influences must be emphasised; prematurity and anoxaemia (asphyxia).

The premature or immature foetus enjoys the advantage of easier delivery due to small size, but this does not compensate for the increased vulnerability of its tissues. Anoxaemia leads to engorgement of the intracranial and visceral venous channels and so makes them more liable to rupture; and in slighter degrees of stress it may give rise to cerebral congestion and oedema.

(c) VARIETIES OF BIRTH INJURY

A complete list of injuries would include almost every tissue and organ in the body. Since intracranial injuries are probably the commonest and certainly the most dangerous, a simple sub-division may be made into (1) cranial and intracranial injuries and (2) other injuries. The latter include some that are dangerous to life e.g. damage to the vertebral column and spinal cord, and rupture of, or haemorrhage into important viscera such as liver, adrenals and spleen; and some that are seldom fatal but nevertheless may require skilled and careful treatment if subsequent morbidity is to be avoided, e.g. fractures of long bones; injury to muscles such as the sternomastoids; and nerve injuries, particularly to the brachial plexus. In the causation of all these extracranial injuries it is noteworthy that obstetrical manoeuvres (e.g. internal version and difficult breech extraction) are mainly responsible.

Cranial and intracranial injuries: The foetal head comes first in 97 per cent. of all births and an understanding of the mechanical problems involved during the passage of the head through the birth passages is essential to the sound practice of obstetrics. There are certain anatomical and physiological features of the foetal skull that serve to protect its contents, and this function is more efficiently performed in a vertex delivery than in a breech. For instance, the skull's plasticity enables it to undergo changes in shape which adapt it to the birth canal; its elasticity enables it to regain its shape in the intervals between stress, so placing the intracranial contents again at rest; its capacity of limited expansion allows respite from the swelling of intracranial congestion and oedema; and the disposition and structure of the dural septa enable them to act as stays or ligaments restraining a dangerous degree of distortion.

In every labour the foetal head is exposed to stress which may be subdivided as follows:—

- (1) a general compression or squeeze of the whole head.
- (2) a simple compression by opposite forces acting at the ends of the long diameter of engagement of the head in the birth-canal.
- (3) "release" effects.

Normal cranial stress leaves no observable effect on the head, or only a moderate amount of distortion (or "moulding") of the cranium and oedema of the scalp which soon disappear. Excessive stress, due to a variety of causes such as narrowing or rigidity of the birth canal, too rapid birth, difficult forceps delivery or prolonged labour, may result in (1) excessive distortion of

the venous sinuses and cerebral veins, producing cerebral congestion and oedema, (2) over-stretching and tearing of the dural septa, sometimes with rupture of certain blood vessels.

The part played during birth by the intracranial dural septa, and the mechanism of dural tears has been clearly described by Eardley Holland (1920). The tentorium cerebelli is more frequently damaged than the falx cerebri, and breech delivery carries the greatest danger. Complete tearing of a septum, severe enough to rupture one of the venous sinuses, is fatal. But haemorrhage may occur within the brain and in the tentorial region before the dural septa are torn; for in cases of marked head moulding and in breech delivery the vein of Galen may be kinked at the point where it enters the straight sinus, causing venous stasis within the tributaries of the vein, and eventually haemorrhage.

Schwartz (1924 and 1927) and Schwartz and Fink (1926) demonstrated tiny haemorrhages and softenings in the brain-substance, as well as pia-arachnoid and tentorial bleedings in two-thirds of all autopsies of babies dying during the neonatal period and in early infancy. Their chief contribution was the demonstration of punctate haemorrhages which were followed later—if the baby survived long enough—by small zones of degeneration and disappearance of cells. When the child's birth had been abnormal these changes were found predominantly in the central white substance of the brain and basal ganglia while cortical damage was relatively infrequent. It seems reasonable to assume, from the distribution of the lesions, that the changes demonstrated by Schwartz were caused mainly by engorgement of the tributaries of the vein of Galen.

Superficial cranial injuries are extremely common. The scalp tissues may show a considerable degree of oedema and haemorrhage (*caput succedaneum*) but this is seldom serious and in most cases it is absorbed remarkably quickly. Sometimes there is an effusion of blood between a cranial bone, often one of the parietals, and its overlying periosteum (*cephalhaematoma*): this again is seldom serious and usually undergoes gradual absorption, disappearing entirely within from six weeks to three months of birth. Occasionally sepsis may supervene, especially when ill-advised attempts have been made to aspirate the blood. Rarely the collection of blood is not entirely absorbed, but undergoes calcification and ossification.

It should be observed that the softness and mobility of the cranial bones protect them from injury except when the stress is very severe. They are seldom fractured except when there has been a difficult forceps delivery, or when some bony projection from the mother's pelvis, such as an osteoma or a beaked sacral promontory, has pressed upon the child's head.

The chief varieties of fractures of cranial bones are:

(1) Depressed fractures. There is deformity of bone without complete loss of continuity and the subjacent intracranial soft parts show little if any damage.

(2) Fractures proper. These may be linear, stellate or comminuted; the parietal bones are especially liable to suffer. There is loss of continuity of bone; both inner and outer layers of periosteum are torn and frequently there is haemorrhage into the subdural space and into the scalp tissues. The effects of this type of fracture are more severe, therefore, than a simple depression of bone and intracranial damage usually occurs.

(d) PATHOLOGY AND PROGNOSIS

When an attempt is made to correlate the experience of obstetricians, pathologists, and paediatricians regarding the part played by intracranial birth injury as a cause of (1) stillbirth (2) neonatal morbidity and mortality and (3) disorders of the nervous system in later childhood it becomes clear that

the subject is one of considerable complexity. It is, however, possible to arrive at certain conclusions which assist in a general understanding of this important subject, and in doing so it is well to avoid too rigid a differentiation of anoxaemia from birth injury, and to accept the fact that when obstetrical conditions favour the probability of trauma to the child, foetal anoxaemia is generally present as well.

It is usual to find one or more of the following pathological lesions when autopsies are performed upon cases of stillbirth and early neonatal death obviously due to severe dystocia:—Gross congestion of intracranial veins and venous sinuses: multiple petechial haemorrhages (with oedema of the brain); ruptures of dural septa; gross haemorrhage (usually venous in origin); fractures of cranial bones. In such cases the causation of death is revealed by the autopsy; and the clinical signs presented by the neonatal cases during the few days before death receive a general explanation, though it is admittedly difficult to correlate individual clinical features with particular pathological changes. It is not unusual to find that a baby injured at birth and showing clinical signs of intracranial damage severe enough to justify a gloomy prognosis surprises the medical attendant by surviving the neonatal period, and the important question of his future health then arises. There is as yet insufficient statistical evidence concerning these cases but all will agree that neurological sequels (mental retardation, epilepsy and, especially, cerebral palsy) are not uncommon in the survivors. It is still not established whether these sequels are more commonly due to gross intracranial pathological changes, such as large haemorrhages, or to minute lesions scattered widely in various parts of the brain. The available clinical evidence seems to point to the following conclusions:—

(1) Hemiplegia (whether unilateral or bilateral) and monoplegia may be due to trauma sustained during birth. In the former cases the arm is more affected than the leg and is held in the position of adduction and flexion; athetosis is common and mental retardation, if present, is secondary to the child's physical incapacity.

(2) Spastic diplegia with symmetrical involvement of the two halves of the body, the legs more affected than the arms, spasticity in excess of paralysis and a considerable degree of mental backwardness are due to prenatal causes, including cerebral maldevelopment and antenatal anoxaemia. The head circumference of these children is generally less than the average and there may be other congenital malformations. Autopsy is likely to show bilateral symmetrical cortical atrophy ("walnut-brain"), due to developmental agenesis, porencephaly, lobar sclerosis or the effects of antenatal haemorrhage (arterial embolism or thrombosis, perhaps with petechial bleedings secondary to antenatal anoxaemia). (Benda, 1945.)

Experienced obstetricians have formed the opinion that children suffering from unilateral or bilateral hemiplegia are more likely to have been born at difficult labours than those who show spastic diplegia, but further statistical evidence is needed on this point and can be obtained only by the co-operation of obstetricians and paediatricians.

In some cases of stillbirth and early neonatal death that at autopsy do not show the more obvious types of injury such as gross haemorrhage, a watery state of the brain with scattered petechiae and generalised intracranial venous congestion may be found; and evidently these morbid changes may be fatal. The clinical signs noted in the neonatal cases of this group bear a close resemblance to those previously described, yet from the point of view of morbid anatomy the damage has been less severe and presumably would have been less likely to give rise to after effects if the child had survived the dangerous early days while acute intracranial disturbance is at its maximum.

In this connection, however, it is important to draw attention to the views of Schreiber (1938 and 1940) and others who reported the finding of multiple areas of "devastation necrosis" in the brain following cerebral asphyxia. Further and more detailed "follow up" studies will be necessary before the frequency of neurological sequelae is known in this clinical group.

The following comments may be made upon regional haemorrhages:

(1) *Bleeding from the veins within the layers of dura mater, and from the venous sinuses*: Subtentorial haemorrhage is likely to be fatal because the vital centres in the medulla are liable to be compressed; this is especially common when septal tears have opened the lateral or straight sinus. Subdural and subarachnoid haemorrhage above the tentorium may be unilateral or bilateral and, if not fatal, may result in compression of the cerebral cortex causing hemiplegia and sometimes epilepsy later in life. Extensive epidural bleeding is rare even when there is a large caput succedaneum.

(2) *Subarachnoid haemorrhage*: This type may have effects analogous to those in adults, causing meningeal irritation and pyrexia; but it may cease spontaneously and much of the extravasated blood is then removed or washed away by the cerebro-spinal fluid. It is unlikely to be fatal unless a major vessel is torn. Pial and cortical petechiae are often associated with this type of bleeding; when excessive or confluent they damage cortical nerve cells and cause paralysis.

(3) *Ventricular haemorrhage*: This is likely to be immediately fatal and it is said to occur more frequently in the premature than in the mature child. Whether the bleeding occurs more often from a choroidal vessel or from an intracerebral haemorrhage tracking into a ventricle is unsettled.

(4) *Intracerebral bleedings*: Haemorrhage due to scattered or confluent petechiae in the substance of the brain, especially when there has been primary or secondary asphyxia, is not incompatible with life; it may result in a central fluid loculation or false porencephaly. Small fluid loculations, either enclosed or communicating with the lateral ventricles have been disclosed by encephalogram and by operation upon children suffering with cerebral paralysis. Scattered petechiae, possibly with associated oedema, may give rise to degenerative changes in fibre tracts connecting cortical ganglia with inferior centres; as a result of this disruption or distortion of pathways, varying degrees of paralysis may arise.

It may be assumed that the two forms of intracranial haemorrhage least likely to cause death are intracerebral petechiae (often caused by primary or secondary anoxaemia) and subarachnoid bleeding.

In all cases of birth injury the immediate prognosis should be guarded during the first three days; survival to the fourth day makes the prospect of recovery more hopeful, though complications such as pulmonary atelectasis and infections (especially pneumonia) may develop. There is no doubt that many of the milder cases and some of the more severe examples recover fully, without any after effects.

(e) PREVENTION AND TREATMENT

Prevention is the ideal. The elimination of contracted pelvis by improving the general health of the population and a reduction of the number of premature births and of conditions leading to antenatal and intranatal asphyxia, would reduce remarkably the incidence of birth injury, especially if women had their first baby when comparatively young. Above all, however, the prevention of birth injury depends upon expert medical and nursing attention during pregnancy and labour; the early recognition of disproportion and of intrauterine asphyxia would enable expert treatment to be given by methods

which involve least risk to the child. Many cases of birth injury are undoubtedly due to lack of obstetrical skill and judgment on the part of the accoucheur, especially when delivery by the breech or by forceps has been necessary. Breech delivery in primigravidae still causes, in many hospitals, a foetal mortality, mainly due to intracranial injury, of 20 per cent. or more. It has been demonstrated that this figure can be lowered to 3 or 5 per cent. by good obstetric technique alone. Lack of care and skill in the application of forceps in the above circumstances is a well known cause of foetal injuries—mainly cranial and intracranial. But this cause has perhaps received undue attention in the past and it is not always recognised that the application of forceps is, in certain conditions, one of the best means of avoiding injury to the child. To use forceps too late, when the child is distressed and asphyxiated and therefore predisposed to intracranial haemorrhage, is just as harmful as their application too soon in labour. A timely episiotomy may serve to prevent fatal intracranial injury in some cases. If birth injuries are to be avoided the special susceptibility of premature babies must be recognised and appropriate steps should be taken.

To summarise, the prevention of birth injury requires that the fundamental principles of obstetrical practice should be applied by doctors and midwives of adequate experience and skill. All concerned should constantly bear in mind the importance of the life and welfare of the child as well as of the mother.

The treatment of the child injured during birth is of great importance and should be in the hands of an experienced paediatrician who can call upon the assistance of suitably trained nurses. Special emphasis should be placed upon the dangers of over-vigorous methods of artificial respiration.

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VI. ASPHYXIA OF THE NEWBORN

In the past it has been customary to think of asphyxia neonatorum as a state of oxygen insufficiency arising during or immediately after birth. It is true that this definition would cover many cases of asphyxia, but a broader conception, which includes consideration of oxygen supply to the foetus before labour, gives a more accurate picture of this important and complicated disorder which probably plays some part in about 50 per cent. of neonatal deaths. Hence has arisen the tendency to replace the term "asphyxia" by "anoxia" or "anoxaemia" and to recognise that a relative insufficiency of oxygen may develop during the antenatal as well as during the natal and neonatal epochs. Furthermore, foetal anoxaemia ("primary" anoxaemia) is sometimes lessened but more often aggravated by the transition from intrauterine to extrauterine life, i.e., neonatal asphyxia may be secondary to foetal anoxaemia; and when the process of birth causes trauma to the child the symptoms of both anoxaemia and birth trauma become closely intermingled.

ANTENATAL ASPHYXIA

(a) Causes

The foetus obtains its oxygen from the mother's blood, and to ensure an adequate supply it is necessary that:—

- (1) The mother's blood should be healthy and adequately charged with oxygen.
- (2) The placenta and umbilical cord should be healthy.
- (3) The foetal cardiovascular system should be normal.
- (4) The foetal blood should be capable of carrying the oxygen and liberating it in the tissues.

We now know, mainly as a result of Barcroft's (1941 and 1942) work, that foetal blood is relatively unsaturated with oxygen when compared with adult blood and to counteract this deficiency the foetal haemoglobin appears to be of a special type and is undoubtedly present in high percentage towards the end of pregnancy. There is also reason to believe that the foetal tissues are, in comparison with those of the adult, capable of tolerating a relatively high degree of anoxia without suffering damage and it has even been suggested that anaerobic metabolism may be possible before birth. An attempt to classify the causes of increased foetal anoxaemia has brought into use such terms as anoxic anoxaemia, when the efficiency of the mother's circulation or respiration is at fault; anaemic anoxaemia, when the mother or child is anaemic; stagnant anoxaemia when the flow of blood in the umbilical cord is impeded; and histiotoxic anoxaemia when the foetal cells (especially of the respiratory centre) are damaged by drugs or disease. In labour the chief causes of foetal asphyxia are antepartum haemorrhage with premature separation of the placenta, prolonged labour or precipitate labour which impedes the placental circulation, undue cranial stress and antenatal asphyxia or anoxaemia.

(b) Effects

The effects of foetal anoxaemia are widespread, and are due not only to lack of oxygen but also to retention of carbon dioxide and other end-products of metabolism; lactic acid accumulates in the tissues and the blood pH. is lowered. These dangerous conditions may be relieved if the child is born speedily and is capable of establishing satisfactory pulmonary respiration without delay. But the time-factor is important because prolonged anoxaemia may damage the foetal tissues, especially the nervous system, cardiac musculature and blood vessels; and the process of birth is potentially more dangerous to the asphyxiated than to the normal child. Furthermore, the effective establishment of pulmonary respiration may be jeopardised by foetal anoxaemia, especially when it has given rise to grave complications such as pulmonary or intracranial haemorrhage.

NEONATAL ASPHYXIA

(a) Causes

The following conditions must obtain if pulmonary respiration is to be successfully established at birth:—

(1) The respiratory centre in the infant's medulla oblongata must have reached an adequate stage of development and must be undamaged, e.g., by haemorrhage, oedema, toxæmia, drug poisoning, etc.

(2) The infant's air passages must be pervious and free from obstruction.

(3) The thorax and upper part of the baby's abdomen must be normally developed and free from disease.

(4) The physiological readjustments which occur during and after birth, not only in the respiratory system but also in other systems, especially the cardiovascular, must take place normally.

(5) The muscular action of inspiration must be sufficiently strong to inflate the lungs.

Hence, in addition to the delayed effects of foetal anoxaemia, the most frequent natal and neonatal causes of asphyxia are prematurity and immaturity; depressed excitability of the respiratory centre due to maternal anaesthesia and sedation; intracranial birth injury; aspiration of blood, mucus, amniotic fluid or meconium into the air passages; and disease or congenital abnormality of the lungs, larynx, trachea and bronchi, thoracic wall and diaphragm.

Barcroft's (*loc. cit*) work upon sheep has shown that rhythmic contractions of the respiratory muscles take place for several months before birth of the foetus and there is little doubt that this is true also for the human infant (Davis and Potter (1946)). Thus the beginning of pulmonary respiration is to be regarded as the culmination of a modulated process, but it is only when birth has occurred that it becomes possible for the infant to achieve the real object of the function, namely the indrawing of air into the respiratory passages; normally this occurs within 30 seconds of birth. It is not to be supposed that even a lusty full time infant can fully expand the lungs by means of a few vigorous inspirations, aided by the effect of forcible expirations through a partially-closed glottis (crying); several days or even weeks may elapse, especially if the child is feeble, during which there is a progressive aeration of the lungs, the expansion of which has been likened by Keith to the opening of a lady's fan. The lungs present a considerable resistance to opening, perhaps mainly due to cohesion between the walls of alveoli; experiments suggest that a pressure equivalent to 20-30 cm. of water is necessary to overcome this resistance (C. A. Smith (1942)) and that there is more resistance to air than to fluid (Gruenwald (1946)).

(b) *Effects*

In fatal cases of asphyxia neonatorum autopsy shows generalised venous congestion and oedema, with dark fluid blood in the veins and petechial haemorrhage into the pleura and pericardium, the meninges, brain, lungs, thymus, heart muscle, kidneys, intestines, etc. Sometimes there is more extensive bleeding, e.g., into the suprarenals, intestines, kidneys, and under the capsule of the liver and spleen. The lungs show atelectasis and in most cases there is, in addition, pulmonary congestion and oedema, with haemorrhage into the lung substance as well as the alveoli. There may be effusion of fluid into the pleural, pericardial and peritoneal cavities. Evidence of birth trauma may, of course, be found in addition; and sometimes it is not possible to say whether an intracranial bleeding is due mainly to birth trauma or to asphyxia. When the child survives, the clinical signs of birth trauma and asphyxia are intermingled.

In the past it has been usual to describe two clinical varieties of asphyxia, namely livida (or cyanotica) and pallida. This elaboration is now considered unnecessary and for practical purposes asphyxia pallida may be regarded as a severe grade of asphyxia livida in which the peripheral arteries become constricted and the rate of circulation is markedly slowed; later there is paralysis of the vaso-motor and other nerve centres.

COMPLICATIONS

In the slighter cases there may be full recovery. Sometimes acute pneumonia develops, especially when the child has inspired infected liquor amnii, blood or meconium during delivery. The persistence of atelectatic areas may give rise to bronchiectasis and chronic fibroid changes (Coryllos (1931); Henderson (1931)).

Whilst it is certain that nerve cells tolerate severe anoxaemia badly and show rapid degenerative changes, it is not yet possible to be dogmatic about the clinical syndromes to which such changes may give rise (see Chapter V, Birth injury). Probably some cases of cerebral diplegia, mental defect and psychopathic disturbance may be caused by cerebral and cerebellar anoxaemia. (Schreiber (1940); Darke (1944); Preston (1945); Rosenfeld and Bradley (1948)).

PREVENTION AND TREATMENT

The prevention of foetal and neonatal anoxaemia is mainly an obstetrical problem and is closely related to the prevention of immaturity and birth injury and to the skilful use of anaesthetics and analgesics for mothers. No subject serves better to emphasise that obstetricians and paediatricians must work in unison if neonatal problems are to be tackled effectively. As noted above, it is probable that asphyxia neonatorum plays a part—and often a leading part—in causing some 50 per cent. of all neonatal deaths; herein lies a strong argument for closer and more skilled observation of both mother and baby during pregnancy and labour. The early recognition and prompt treatment of intranatal asphyxia is of first importance.

The treatment of the baby born in a state of asphyxia need not be dealt with here, except to draw attention to the essential points.

1. *General care of the child*

The child should be handled gently, kept at a normal temperature and allowed to remain as quiet as possible.

2. *Clearing of the airways*

This is important but it should be carried out gently, and preferably by suction through a non-metal catheter. The child should be placed on its side with head lowered.

3. Aiding the initial inspiratory effort

The administration of oxygen, or of 5 per cent. carbon dioxide in oxygen, is valuable; but the pressure should be controlled and for this a special apparatus is desirable.

Intermittent gentle compression of the chest is permissible; the old fashioned vigorous methods of attempting to promote respiration are dangerous as well as futile. Various respiratory and cardiac stimulants can be used but, on the whole, the results are disappointing. Once respiration is established the child's essential requirement is oxygen.

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VII. MEDICAL AND NURSING CARE OF THE NORMAL NEWLY BORN INFANT, INCLUDING BREAST FEEDING

To preserve good health in the newborn infant there must be efficient nursing care and due attention should be paid to the following essential requirements:—(a) an adequate supply of human milk; (b) gentleness in handling and avoidance of meddlesome interference; (c) supervision of respiration and temperature; and (d) protection from infections. It cannot be emphasised too strongly that certain details of nursing technique, such as

the cleansing of the skin, eyelids, mouth and nose may cause injury unless they are carried out very carefully and with due regard to the delicacy of the baby's tissues.

As soon as possible after birth the baby should be wrapped in warm, sterile blankets and placed in a room adequately heated and of reasonable humidity—e.g., temperature 70°F. and humidity 55 per cent. The child's temperature at birth is usually about 1°F. higher than that of the mother; subsequently it tends to conform to that of the surroundings. In many homes there is a tendency to overclothe the baby in a room which is too hot and of insufficient humidity.

Following the onset of satisfactory pulmonary respiration a preliminary cleansing of the skin may take place. The vernix is insoluble in water but can be readily removed by sterile olive oil. Soap and water in a modified blanket-bath may be used and then light massage of the skin followed by powdering is beneficial because it usually induces a good bout of crying which helps to expand the lungs. Great care should be taken to avoid any injury to the delicate cuticle of the skin, especially in the axillae and groins.

The cutting of the umbilical cord makes a potential portal of entry for microorganisms which may pass directly into the baby's blood stream, or into the peritoneal cavity, or may lodge in the liver. Careful attention to asepsis is therefore necessary in ligaturing, cutting and dressing of the cord. It is important to keep the cord dry and a desiccating powder such as sterilized talcum and zinc oxide in the proportion of 3 to 1 should be applied fairly liberally. The dressing is kept in place by a light woollen binder applied loosely in order that respiration may not be impeded. After the cord stump falls away spontaneously about the fourth or fifth day, the granulating surface should be protected by a sterile dressing until it is covered by permanent scar tissue.

It is probable that in the past the nursing care given to the baby's eyes and nose has been over-zealous; any blood, vernix or mucus should be removed very gently by cotton wool swabs soaked in boracic lotion—nasal obstruction which cannot be removed in this way can be overcome by introducing a small soft sterile rubber catheter which has been well lubricated with sterile liquid paraffin. Bacterial invasion of the naso-pharynx in the first few days may cause a nasal discharge which should not be confused with that of syphilitic rhinitis.

It is doubtful whether antiseptic eye drops should be instilled as a routine for all newly born babies; but when there is suspicion of maternal gonorrhoea or of trauma to the baby's eyelids during birth freshly prepared drops of silver nitrate ($\frac{1}{2}$ per cent.) may be used, or alternatively systemic administration of penicillin started at once after birth.

BREAST FEEDING

Breast milk is the food designed by nature for the human infant. This single fact is a very strong argument in its favour as against artificial feeding. But, in addition, it may be pointed out that breast feeding is cleaner, cheaper and when lactation is satisfactorily established, easier than artificial feeding. Furthermore, the general progress of breast fed infants is more satisfactory than that of artificially fed babies; digestive upsets are less frequent and all forms of infection are fewer and less dangerous. Impressive figures supporting these statements have been furnished by Feldman (1927) and by Grulee, Sanford and Herron (1934): the former found the infant mortality in breast-fed infants to be 22.9 per thousand, as against 108.3 per thousand in

artificially fed infants; the latter, in a study of over 20,000 babies, found the infant mortality rates to be 6.7 and 27.2 per thousand respectively. It must be remembered, however, that any group of artificially-fed babies will be likely to include a number who have made unsatisfactory progress on breast-feeding.

The danger of infecting an artificially fed child is considerable unless (1) the milk is of good quality and has been rendered free from bacterial infection by pasteurisation, boiling or drying; (2) feeding bottles, teats and utensils are carefully sterilized; and (3) the hands of the mother or nurse preparing the feeds are free from infection.

Not every mother is able to breast feed her baby; but many mothers fail to do so when they could. There are some mothers for whom breast feeding is contraindicated on medical grounds and others for whom it is a physical impossibility; but it is probably true to say that 80 to 85 per cent. of all mothers should be able to breast feed their babies. The prospects for successful breast-feeding are much improved when the mother is adequately nourished and lives in good environmental conditions; when her hygiene and psychological circumstances are normal, and when she understands the technique of breast feeding and is confident.

Recent studies in America (Macy and others (1945); Lawrence and others (1945)) give the following figures for the vitamin content of fresh mature human and cow's milk.

Vitamin concentration per 100 ccs.	Human milk	Cow's milk
Vitamin A. International units	360	183
Vitamin D. International units	0.4 to 10	0.5 to 4
Vitamin C, milligrams	5	2
Thiamine, micrograms	14	38
Riboflavin, micrograms	37	200
Nicotinic acid, micrograms	183	85
Pantothenic acid, micrograms	246	350
Pyridoxin, micrograms	4	67
Biotin, micrograms	0.8	3

Neither the vitamin D nor the vitamin C content of breast milk is adequate to ensure protection against rickets and scurvy respectively and these vitamins should be added to breast-fed infants' diets as well as to those of artificially-fed infants.

Normal breast milk is not entirely free from potentially harmful bacteria. Investigations by Duncan and Walker (1942) and by A. W. Downie (1942), have shown that many specimens of breast milk contain staphylococci (both albus and aureus) though there are no clinical signs of mastitis; the presence of these organisms should not be regarded as a contraindication to breast feeding. There is still much to be learnt about milk-borne infections, the technique of storing breast milk, the development of immune bodies by the baby and many other related problems.

Technique of breast feeding

Regularity of feeding and careful observation of the baby's progress are certainly desirable, but attention has been rightly called to the harm that may be done by adhering to a routine that is too strict and stereotyped. This is especially liable to happen in institutions, and if the babies are weighed too often there is likely to be a tendency to resort too readily to artificial feeds.

The infant should be nursed in a comfortable position and this is best accomplished with the mother sitting—even early in the puerperium she can be propped up during the feeding times. The duration of the feeding time will vary according to the individual infant, but since the bulk of the feed is taken in the first five minutes, a total of twenty minutes is usually sufficient for most babies. After about ten minutes of feeding the infant should be taken from the breast for a few minutes and held in an upright position so that swallowed air may be belched; and this manoeuvre should be repeated at the end of the feeding. The interval between feeds may be either three hours or four hours; in either case regularity is desirable in the early days. A four hourly schedule is generally preferable but small infants and those showing a slow gain in weight may benefit from three hourly feeding.

During the first 48 hours after birth the baby may be allowed not more than five minutes at each breast; but subsequently, when the likelihood of damage to the nipples is less, the time may be prolonged to ten minutes.

A baby's nutritional needs will usually be satisfied by a daily intake of $2\frac{1}{2}$ ozs. of breast milk per lb. of his body weight; thus an 8 lb. baby would need 1 pint in 24 hours. His gain in weight should be 6 to 8 ozs. per week and he should be happy and contented.

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VIII. PREMATURITY AND IMMATURITY

It is not yet possible to evaluate fully the effects of prematurity and immaturity on the stillbirth and neonatal mortality rates and on neonatal morbidity. Only recently has there been any system of certification of prematurity and the state of maturity or immaturity is often omitted from death certificates when death has been ascribed to birth injury, asphyxia, atelectasis, congenital malformation or neonatal infection. In many cases the weight of stillborn infants is not recorded and knowledge of their state of maturity is similarly lacking.

Maternity hospitals' reports may provide accurate details on these matters but their clinical material is to some extent selected and their records cannot be considered applicable to the community as a whole. Information of a general character about the incidence and dangers of prematurity is, however, available.

(a) DEFINITION OF PREMATURITY AND IMMATURITY

In accordance with the standard in international use any infant whose birth weight is $5\frac{1}{2}$ lb. (2,500 grammes) or less is considered either immature or premature according as the estimated period of gestation is full time or less.

Henderson (1945) has suggested a minimal weight standard of 2 lb. 12 oz. (1,250 grammes), the average foetal weight at 28 weeks, and any infant below this weight would be classified as "pre-viable".

(b) INCIDENCE OF IMMATURITY

In statistics obtained from hospitals, premature births vary between 6 per cent. and 18 per cent. of the total births, but the admission of abnormal obstetrical cases requiring special observation and treatment naturally causes the percentage to be higher in a maternity hospital than it is in a large district. Crosse (1945) who analysed the figures for the City of Birmingham in 1943 found that of 19,912 total births, 1,268 were immature (i.e., 6.3 per cent. of total births, living and still). Of the 536 stillbirths 222 (41.4 per cent.) were prematurely born.

Analysis of 8,808 consecutive "booked" cases admitted to the Aberdeen Maternity Hospital (Baird, 1945) showed that labour terminated prematurely in 738 cases (8.38 per cent.).

In some sections of the community—e.g., normal multiparae who are in favourable circumstances—the incidence of prematurity may be as low as 2 per cent. of live births, or even less; Crosse (1945) reported that the incidence was 4 per cent. in approximately 11,500 deliveries which took place in the patients' own homes. Of the 674,780 notified (as distinct from registered) live births in England and Wales in 1945 there were 30,173 infants (4.5 per cent.) who weighed $5\frac{1}{2}$ lbs. or less at birth, and of that number 36.8 per cent. were born at home and 63.2 per cent. in hospital. (Report of the Ministry of Health 1947.)

(c) DANGERS OF IMMATURITY

Infants who are born prematurely and are immature for separate existence are in special danger both during and after birth. The fragility of their tissues renders them liable to damage during delivery and their organs have not reached a sufficiently advanced stage of development to allow them to undertake their new functions satisfactorily. Hence the vasomotor and respiratory centres respond inadequately and there is a liability to cyanosis, oedema, asphyxia, pulmonary atelectasis and pneumonia; the heat-regulating centre is unable to ensure that the body preserves a steady temperature. The

ingestion and digestion of milk are imperfect and there is a danger that the milk may be aspirated into the respiratory tract. The defence mechanisms against microorganisms, being less effective than those of the mature infant, are liable to break down. Other handicaps from which the immature baby suffers include imperfect renal function (Young, Hallum and McCance (1941), Young and McCance (1943)), incomplete development of the endocrine system and lack of stored iron.

A majority of the very small immature babies die simply because they are functionally incapable of separate existence; but if these infants are excluded there seems to be little doubt that the prime cause of stillbirth and death of premature babies within the first few days is birth injury—usually intracranial in site. Illingworth (1939) in his series of 150 prematures and 150 controls, found the incidence of cerebral damage to be: prematures 20 per cent., controls 6.66 per cent. Hess, Mohr and Bartelme (1934) found that the incidence of intracranial haemorrhage in their series of 250 prematures was 69 (i.e., 27.6 per cent.); whilst Capper (1928) found that death of the premature infant during the first week of life was very frequently due to intracranial haemorrhage and that the more immature the infant, the higher the incidence of such injury.

Apart from intracranial injury, the other common cause of death of the premature baby within the first 48 hours is asphyxia. After the first week of life, however, the neonatal mortality amongst prematures and immatures is more often due to infection—particularly of the lungs, intestinal tract and skin.

It is not, therefore, surprising that immaturity is directly or indirectly responsible for a high proportion of neonatal deaths and disease, though it is often difficult to decide whether the immaturity has been the lethal factor or only a predisposing one.

The Statistical Review of the Registrar-General for England and Wales states that during the year 1938 there were 17,572 neonatal deaths of which 8,984 (slightly over 50 per cent.) were certified as due to prematurity—i.e., 14.47 deaths per 1,000 live births; and it is probable that about 50 per cent. of the 24,729 stillbirths in the same year were also associated with prematurity. This would constitute a total of about 21,000 infant deaths associated with immaturity or 33 per 1,000 total births. Sandifer (1944) points out that this total was almost as large as the deaths certified as due to "tuberculosis of all forms" during the same year. In 1945 there were 16,910 neonatal deaths in England and Wales, and of these 7,116 were certified as due to prematurity; i.e., 10.4 deaths per 1,000 live births. If it be assumed that about 50 per cent. of the 19,333 stillbirths in the same year were associated with prematurity, the total infant deaths due to or associated with prematurity is 16,782 or 24 per 1,000 total births.

The mortality rate of immature babies per 1,000 live births varies from district to district; in Greater London in 1938 the figure was 11.47, in South Wales 16.41 and in Durham and Northumberland 17.17.

(d) AETIOLOGY OF PREMATURETY

Analysis of groups of premature births shows that a cause of prematurity can be demonstrated in only about 50 per cent. of cases; toxæmia of pregnancy, antepartum haemorrhage, multiple pregnancy and foetal malformation are mainly responsible. For instance, out of 113 cases of known aetiology studied by Falkiner (1943) at the Rotunda Hospital, Dublin, 24 were ascribed to multiple pregnancy, 40 to toxæmia of pregnancy and 19 to antepartum haemorrhage; the other cases were listed as follows: Foetal

abnormality 7; various maternal conditions (e.g., cardiac and pulmonary disease) 6; induction for disproportion 6; syphilis 4; pyelitis 4; severe anaemia 3.

A similar study of 298 premature deliveries of known aetiology at Aberdeen Maternity Hospital (Baird (1943)) revealed the following causes:

	<i>Cases</i>
Pre-eclampsia	102
Twin pregnancy	102
Gross foetal deformity	20
Accidental haemorrhage	20
Miscellaneous	18
Syphilis	12
Placenta praevia	10
Heart disease	9
Pyelitis	5

Sandifer (1944) in a communication dealing with 1,000 cases of premature birth at Queen Charlotte's Maternity Hospital employed the following classification:

- (a) Cause unknown 372 cases (37.2%)
- (b) Premature birth (labour induced) 319 „ (31.9%)
- (c) Premature birth (labour not induced) ... 309 „ (30.9%)

He analysed the causes of spontaneous onset of premature labour and the indications for induction of premature labour with the following results:

	<i>Cases</i>
Toxaemia of pregnancy, including nephritis and hypertension	274
Multiple pregnancy	178
Accidental antepartum haemorrhage	48
Placenta praevia	43
Foetal malformation	32
Disproportion (labour induced)	16
Hydramnios without foetal malformation	12
Pyelitis of pregnancy	8
Heart disease	3
Syphilis	1
Sundry maternal conditions	8
Sundry foetal conditions	5

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He emphasizes the need for caution in reaching conclusions as to the aetiology of premature labour in any one case. The finding of some maternal disease does not necessarily mean that it is a causal factor—it may be coincidental. For instance he found that heart disease, which is often stated to be a cause of prematurity, was commoner among patients giving birth to mature babies, than among those having premature babies.

(e) PROGNOSIS

The chances of survival after premature delivery vary with many factors, the most important being the degree of immaturity and this is best measured by considering the period of gestation and the infant's birth weight.

The following figures demonstrate this point:—

Holt and McIntosh (1940)

<i>Birth Weight</i>				<i>Survival Rate</i>
1,000	grammes (approx. 2 lbs.)	...	...	5 per cent.
1,000-1,500	" (" 2-3 lbs.)	...	...	33 " "
1,500-2,000	" (" 3-4 lbs.)	...	...	75 " "
2,000-2,500	" (" 4½-5½ lbs.)	...	...	90 " "

Crosse (1945)

Weight		No. of infants admitted (1931-42)	Percentage sent out alive and weighing over 5 lbs.
0-2 lbs.	0- 909 grammes ...	79	3
2-3 lbs.	909-1,364 grammes ...	347	27
3-4 lbs.	1,364-1,819 grammes ...	832	60
4-5 lbs.	1,819-2,274 grammes ...	865	78
5-5½ lbs.	2,274-2,500 grammes ...	209	94

Sandifer (1944)—(Queen Charlotte's Maternity Hospital 1936 to 1942).

Birth weight		No. of children	Alive on discharge	Died in hospital	Stillbirths	Natal and neonatal death rate
						Per cent.
5-5½ lbs.	...	309	246	20	43	20.4
4½-5 lbs.	...	211	167	11	33	20.8
4-4½ lbs.	...	155	104	22	29	44.4
3½-4 lbs.	...	118	51	31	36	56.8
3-3½ lbs.	...	88	25	27	36	71.6
2½-3 lbs.	...	55	9	15	31	83.7
2-2½ lbs.	...	41	2	15	24	95.1
Under 2 lbs.	...	23	0	3	20	100.0

The report by Asher (1946) contains evidence in general agreement with the above figures.

In hospital records the mortality rate of premature infants varies between 100 and 350 per 1,000 infants born prematurely, and for comparison it may be noted that the corresponding death rate for mature infants varies from 7 to 25 per 1,000. Again, quoting Crosse's report based upon the figures for the City of Birmingham the neonatal mortality rate for immature infants for 1943 was 278 per 1,000, while for mature infants it was slightly over 11 per 1,000. Approximately 50 per cent. of the total stillbirths and neonatal deaths were associated with prematurity, and this figure is generally accepted as representative of the country as a whole. The added handicap of possible morbidity in survivors should not be forgotten. In certain centres where special efforts have been made to establish the best possible conditions for the care of the immature child the mortality rate has been brought down to levels which, in comparison with usual standards, are commendably low, e.g., Field (1943) in London 8.1 per cent.; Crosse (1945) in Birmingham 12.8 per cent. These figures should not necessarily be taken to indicate the relative efficiency of the centres quoted, because there are individual factors, e.g., the relative proportion of infants of very small birth weight, which make the groups not strictly comparable; but they are an encouraging indication of what can be achieved by special endeavour and enthusiasm.

Approximately two-thirds of deaths in prematurely born children occur within 48 hours of birth and include most of the infants of very small weight. This fact is exemplified in the following table: (J. H. Hess (1947)).

MORTALITY RATES OF PREMATURE INFANTS OF THE SARAH MORRIS HOSPITAL
BETWEEN YEARS 1922-1940, BASED ON WEIGHT AND TIME OF DEATH

Birth Weight in grammes	Total admis- sions	Number gradu- ated	Deceased first 24 hours	Deceased 24-48 hours	Deceased after 48 hours	Survived, per cent.
Less than 1,000 ...	247	32	136	28	51	12.95
1,001-1,250 ...	286	110	89	34	53	38.46
1,251-1,500 ...	465	266	97	28	74	57.20
1,501-2,000 ...	1,391	1,090	113	37	151	78.36
2,001-2,500 ...	1,359	1,209	53	22	75	88.96
Over 2,500 ...	135	123	5	1	6	91.11
Total ...	3,883	2,830	493	150	410	72.88

Hence it may be assumed that after the first two days there is a greatly improved prospect of survival (Holt and McIntosh (1940)). In a study of 921 premature infants dying in the neonatal period, Graham (1935) found that 52.8 per cent. died in the first 24 hours, 13 per cent. in the second 24 hours and 34.2 per cent. in the later neonatal period.

Birth injury (either intracranial or extracranial), asphyxia, antenatal disease (e.g. syphilis) and deformity operate adversely upon the immature infant. Factors such as the availability of breast milk, the standard of medical and nursing care provided, the housing and other environmental conditions and the threat of infective processes all play a considerable part in the prognosis.

The importance of breast feeding for premature babies is emphasised by Crosse (1945) who followed up those babies discharged home from hospital as healthy. She found at the end of one year a 4 per cent. mortality among those who had been breast fed for at least 6 months, as against 10 per cent. among those which had been fed entirely artificially.

(1) *Physical Development.* The subsequent physical development of babies born prematurely has been investigated by Illingworth (1939), Collis and Majekodunmi (1943), Hess, Mohr and Bartelme (1934), Capper (1928), and Asher (1946). The findings are not all in agreement but it seems that many premature babies attain average height by the time they are three years old; but a higher proportion, especially boys, remain somewhat under weight up to puberty. Factors influencing the physical development of a premature infant are similar to those influencing its chance of survival; thus, the lower the birth weight, the longer it takes to attain normal height and weight; breast-fed babies reach average height and weight more quickly than those that are fed artificially.

(2) *Mental Development.* The subsequent mental development of babies born prematurely has been investigated by Hess, Mohr and Bartelme (1934), Osselmann (1930), Gries (1931), Sarvan (1930), Capper (1928), Brander (1937), Holt and McIntosh (1940) and Asher (1946). Again there is no uniformity of opinion. Some claim that there is no significant difference in the intelligence of the infant born prematurely as compared with the normal infant; others conclude that premature children are less intelligent and tend to be backward at school or mentally abnormal in other ways. Evidence has been produced suggesting that maternal dependency, lack of self reliance, bad temper and lack of friendliness to other children are all found more commonly among those born prematurely.

Nevertheless, many men famous for their mental ability as scientists, statesmen, politicians and writers are reputed to have been premature or immature at birth.

The ultimate outlook for premature babies is a subject that requires further study before definite conclusions can be stated. There is good reason to believe, however, that premature babies, if free from congenital stigmata and inherited disease and if given special care, will usually grow up normally. Without special care the premature child advances more slowly, both physically and mentally, than the mature baby.

The diseases to which immature infants are specially liable are as follows:

(a) *Cerebral damage or abnormality*

The susceptibility of premature babies to intracranial birth injury has already been emphasised. Those that do not die at birth are therefore liable to the after-effects which have already been described.

(b) *Anaemia*

There is some suggestion (Lichtenstein (1921)) that the haemoglobin level at three months of age is lower in babies born prematurely than in mature babies. The findings of Lichtenstein were not confirmed, however, by Mackay (1935); but there is little doubt that the premature baby, deprived of the iron storage which occurs mainly in the later weeks of intrauterine life, is prone to anaemia.

(c) *Infection*

Although premature babies are specially susceptible to infection—particularly of the respiratory tract—during the neonatal period, there does not appear to be any particular tendency in later life to specific infectious fevers. (Illingworth (1939)).

(d) *Malnutrition and Rickets*

The ingestion, digestion and absorption of milk are imperfect in the premature baby and handicaps of this kind appear to continue for some considerable time after birth; hence there is an increased tendency to malnutrition and rickets unless special preventive measures are taken.

(f) **PREVENTION AND TREATMENT**

Measures to reduce the frequency of premature labours are of the utmost importance, but the prospects of successful prophylaxis are naturally dependent upon the aetiology of each case, and it must be realised, as previously stated, that the cause remains unknown in about 40 per cent. to 50 per cent. of all cases—even when the mothers have received antenatal care at clinics staffed by obstetricians. There is, however, some reason to believe that these unknown causes will become fewer as preventive treatment of a general character is extended—e.g. improvement in economic, social and educational status of the poorer classes (see Chapter IV). The Report by the Department of Health for Scotland on Infant Mortality in Scotland (1943) illustrates that the prematurity rate is much less in the upper middle class women than in women of the “hospital” class and the incidence of pre-eclampsia is also lower. Upper middle class patients, corresponding to the Registrar General’s classes I and II, are better developed and nourished than “hospital patients” in the same area. Such feeding experiments as have been carried out suggest that the incidence of prematurity can be lowered by suitable diet. It is unlikely, however, that even first class nutrition during pregnancy will entirely neutralise the effect of years of faulty diet dating from early childhood.

It is doubtful if overwork is an important factor in causing premature labour, for the incidence of prematurity is just as high in primigravidae as in multiparae.

Apart from efforts to improve the standards of general health during pregnancy, chief hope would appear to lie in reducing the incidence of toxæmia of pregnancy and of accidental antepartum hæmorrhage. There seems to be little prospect, at present, of reducing the incidence either of foetal malformations or placenta prævia, but modifications in the treatment of the latter may well reduce the number of cases of prematurity associated with it.

When twin pregnancy is diagnosed the risk of premature onset of labour is so great that the patient should be admitted to hospital during the later weeks of pregnancy for special care and rest if these cannot be obtained at home.

The *treatment* of immature babies presents considerable difficulty. First and foremost, the child should be in the care of specially trained nurses, working under the supervision of a paediatrician who has had experience of newly born (including immature) babies; secondly, the child's environmental conditions should be satisfactory; thirdly, an adequate supply of breast milk should be available. It is incumbent upon every district to plan and administer a suitable scheme for the care of immature babies, and attention should be drawn to the programmes that have been put into operation by certain of the American States (Hess and Lundeen 1941).

While it is possible to rear the bigger immature babies in their own homes, especially when the parents are not of "low income" status, the outlook for the child would be greatly improved if accommodation were provided in a special "premature station" with adequate staff and equipment (Hess (1947)). Ideally this premature station should be situated in the grounds of the medical teaching centre and in immediate proximity to the maternity and children's hospitals. The babies should be nursed in separate chambers or cells (British Paediatric Association Report (1943)) which have special arrangements for conditioning the air in regard both to temperature and humidity and some of the cots should be of special type. The number of cubicles required will depend upon the type of obstetrical work done in the hospital and the size of the associated "district." In those maternity hospitals which deal with a relatively high proportion of abnormal cases the prematures may be 10 per cent. to 20 per cent. of the total infants; in hospitals which accept a higher proportion of normal cases the prematures will be 5 per cent. to 10 per cent. of the total infants. When a baby is admitted to the premature station from the "district," accommodation should be available for the mother also.

(g) MEDICAL AND NURSING CARE OF PREMATURE AND IMMATURE INFANTS

Immediately after birth the immature child should be wrapped in cotton wool, placed in a warm cot and left undisturbed but under close observation for eight hours. The child's head should be a little lower than the body during the first twelve to twenty four hours, and subsequently whenever an attack of cyanosis or collapse occurs. After the first day the head should be slightly raised, particularly during and after feeds. Shallow breathing is an indication for administering warmed 5 per cent. carbon dioxide in oxygen by nasal catheter or by a special rubber mask; but it is better to place the child in a chamber filled with carbon dioxide-oxygen mixture (Clifford (1947)). When the baby is cyanosed oxygen should be administered continuously; for this purpose it is desirable that there should be a piped supply of oxygen.

After an interval of 8 to 12 hours the baby's skin may be cleansed very gently with soap and water but no attempt should be made to remove all the vernix lest injury be done to the delicate cuticle. Oiling the skin with sterile olive oil will help to prevent dryness and cracking. For the smaller immatures a gamgee jacket, and cotton wool around the limbs and as a cap over the head will prevent loss of heat; the bigger babies may be clothed in a light loose gown of cotton and silk mixture covered by a woollen jacket fastened with tapes down the front. Soft stockings and gloves may be worn. Gauze or absorbent wool may be used instead of a napkin. The binder (crêpe bandage) to keep the umbilical dressing in place should be wrapped around the infant so loosely that there is no compression of the abdomen even when a feed has been taken. The doctor will have to decide upon the frequency of weighing and temperature-taking in each case, bearing in mind the importance of avoiding any unnecessary interference. It is customary to take the temperature in the rectum, using a low-reading thermometer and charting the actual temperature recorded; a fairly constant rectal temperature of 98-99°F. is the ideal.

The temperature of the cubicle or room in which the baby is nursed should be maintained between 70°F. and 80°F., and double doors will be necessary. In a premature station it is desirable to have various sections at graded temperatures in order to accustom the infants gradually to normal conditions. The desirable degree of humidity lies between 55 per cent. and 65 per cent. of saturation.

For the provision of warmth hot water bottles may be slung on the sides of the cot or a light electrically heated blanket may be used; the bed-temperature is kept at 80°F. to 95°F. according to the weight of the baby. The child should be protected from the weight of the bed clothes by the use of a wire cage. The clothing should be loose in order that the respiratory movements of the chest and abdomen may not be impeded, and the baby's position should be changed frequently to encourage full expansion of the lungs. Nurses, doctors and all other persons who come into close contact with the child should be masked and gowned and it should be handled as little as possible.

There may be difficulty in the feeding of the premature child because the infant's sucking and swallowing reflexes are feeble; furthermore, inhalation of milk, or of vomited material is liable to cause asphyxial attacks with cyanosis and the danger of circulatory failure. It is therefore necessary to give small feeds at frequent intervals; the capacity of the baby's stomach is small and overloading may cause respiratory embarrassment and intolerance for food. When the infant is dehydrated, 5 per cent. glucose in sterile tap water may be offered; and if there is vomiting it may be necessary to give 5 per cent. glucose in one-fifth normal saline intravenously. Occasionally blood plasma with 10 per cent. glucose solution in equal parts may be a useful transfusion, or dried plasma dissolved in 10 per cent. glucose instead of water.

The volume of breast milk given at each feed varies with the weight of the baby and its vitality; for instance one, two or three drachms two hourly (10 feeds in 24 hours) may be given in the early days with a gradual progressive increase to the point when the child is receiving 2 to 2½ ozs. of fluid per lb. of body weight in 24 hours. The volume of milk taken must be charted carefully. Though the child may eventually require a diet which gives as much as 60-90 calories per lb. of body weight daily, overfeeding of the immature baby is liable to cause asphyxia or diarrhoea; therefore the caloric intake during the early days should not be more than 35 to 40 per lb. of body weight daily.

Very small immature babies are best fed lying in their cots, giving expressed breast milk with a pipette or Breck feeder. Stomach-tube feeding is invaluable when the baby is too feeble to swallow, but special care must be taken to pinch the tube firmly before it is withdrawn, in order to prevent the spilling of milk into the child's pharynx and larynx.

(When the baby reaches a weight of $4\frac{1}{2}$ -5 lbs. it can usually take feeds directly from the breast.

Occasionally immature babies require a feed of higher caloric value than breast milk if they are to gain weight satisfactorily and various formulae have been used, e.g., the addition of calcium caseinate with added sugar, or dried lactic acid milk to breast milk or the use of evaporated cows' milk in appropriate dilution. The most recent suggestion, and undoubtedly the most promising, is the addition of casein hydrolysate to breast milk; the quantity at present recommended varies between $1\frac{1}{4}$ and $2\frac{1}{2}$ drachms in 10 ozs. of milk feed.

When breast milk is not available a "humanised" dried milk may be used at half strength, with gradual increase of concentration.

Medical treatment takes secondary place in comparison with feeding and general nursing care; but many have claimed that thyroid extract (gr. $1/20$ per lb. of body weight) is helpful during the early weeks; and nikethamide (0.5 to 1.0 cc. intra-muscularly) may be used in addition to oxygen when respiratory and vasomotor collapse threatens. Vitamins are best given in concentrated form; the daily requirements would appear to be approximately as follows: vitamin A, 3,000 units; vitamin D, 1,400 units; vitamin C, 50-100 mgms. Recently it has been claimed that larger doses of vitamin C are helpful if the child is receiving a high protein diet. Some writers recommend that vitamin K by mouth be administered to all immature babies, but agreement has not been reached on this point.

Iron in the form of crystalline ferrous sulphate beginning with $\frac{1}{2}$ gr. daily may be started after the fourth week, and the dose should be gradually increased to $2\frac{1}{2}$ -3 grs. daily at the age of six months.

Nursing Staff

The nurses must be adequate in number (day and night) and no nurse should be responsible for more than two infants. The seniors should hold the Certificate of Sick Children's Nurses and should have had special training with immature babies; they should not be employed in other duties. Pupil midwives should receive instruction in the nursing of prematures. One of the main functions of the premature nursery is to improve the training of nursing personnel and stimulate their interest (Bloxsom (1946)).

Feeding of Infants

A supply of breast milk is so important to immature infants that a breast milk bank should be set up in order to supplement the milk available from the other mothers in the hospital, and to provide when necessary for the feeding of prematures who have to be nursed in their own homes. (Crosse (1945); Dynsky-Klein (1946)).

Danger of Infection

All the recommendations in the section dealing with neonatal infections should be carried out with special care for immature babies.

Transport of Immature Babies

A specially heated and equipped ambulance should be available to ensure that a patient born at home may be transferred speedily and with as little danger as possible to the premature station.

Discharge from Hospital of Immature and Premature Babies

Care is necessary when babies are discharged from a premature station to their homes. By that time they should have attained a weight not less than 5 lbs., and they should no longer require special treatment. Steps should be taken to ensure that the home conditions are satisfactory, and that the mother is fully instructed in the care of her child. Further supervision by means of health visitors, and by attendance at an infant welfare clinic is desirable.

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IX. DISEASES OF THE NEWBORN

It will not be necessary to describe all the diseases to which the newborn baby is liable. The various infections, constituting the most important group, are dealt with on page 62, but reference must also be made to neonatal haemorrhage, anaemia and haemolytic disease of the newborn.

(a) NEONATAL HAEMORRHAGE

The dangers of haemorrhage during the neonatal period are twofold; firstly, newborn infants tolerate loss of body fluids (especially blood) badly; secondly, when visceral bleedings occur the functions of the affected organs or tissues are damaged and thus an additional handicap is imposed.

The more common types of haemorrhage met with in the neonatal period are:—

(1) Those resulting from birth injury, e.g. superficial injuries of the scalp and skin elsewhere; lacerations of mucous membranes; intracranial and visceral haemorrhages.

(2) Those due to asphyxia.

(3) Those secondary to general or local disease, e.g. syphilis, neonatal infections and septicaemia.

(4) Uterine haemorrhage, which may be pathological but is usually physiological ("genital crisis of the newborn") and due to the withdrawal of the hormonal influence of the placenta.

(5) Haemorrhagic disease of the newborn.

(6) Umbilical bleeding.

(7) Those due to certain rare blood diseases, e.g. haemophilia, congenital thrombocytopenia, and congenital fibrinogenopenia.

There is general agreement that from the second to the seventh day after birth the prothrombin level in the blood plasma is low and the coagulation time of blood is relatively prolonged. The discovery of vitamin K, which plays an important part in the synthesis of prothrombin has solved some of

the perplexities surrounding neonatal haemorrhage, but many questions still await answers. Investigations upon newborn babies indicate that spontaneous haemorrhage seldom occurs, unless the level of prothrombin is conspicuously low, e.g. about 20 per cent. of the average adult level, but there does not appear to be a proportional relationship between the fall of prothrombin and the occurrence and severity of the bleeding. Indeed, Scobbie (1942) found a prothrombin index of less than 50 (normal figure 100) in apparently healthy infants, while bleeding occurred with indices above 50 in five out of fifteen cases.

It has been suggested that the foetus is unable to store vitamin K and that this lack of it is not overcome until bacterial synthesis in the infant's intestine has been made possible by milk feeds. It certainly appears to be established that the sooner milk, especially cow's milk, enters the child's bowel, the more quickly is the prothrombin level raised. A diet containing food rich in vitamin K does not seem to help the mother in safeguarding her infant against a fall of prothrombin after birth (Kove and Siegel (1940)); but this fall is definitely prevented when the mother is given vitamin K therapeutically within a few hours of delivery or when the baby is given vitamin K (Lehmann (1944)).

Undoubtedly the factors concerned in neonatal haemorrhage are numerous, including prothrombin content, physical trauma to vessel walls, e.g. birth trauma and asphyxia, and possibly nervous and biochemical influences which interfere with the contraction of capillaries. Even though there is still much to be discovered about the factors concerned and their mode of action, it is reasonable to take such therapeutic steps as may be possible to counter the fall of the prothrombin which commonly occurs and there is evidence that by such action the incidence of bleeding during the neonatal period is lessened. Hence the wisdom of administering vitamin K to the mother if labour is likely to be abnormal or the baby likely to be immature. (Macpherson *et al*, (1940)).

Haemorrhagic Disease of the Newborn

Under this heading may be grouped all examples of spontaneous neonatal haemorrhage which occur without such obvious cause as birth injury, gross asphyxia, infection or definite disease. It is dramatic and dangerous and it affects the sexes equally; its frequency seems to vary between 1 in 600 and 1 in 200 of all births. In 1945, 257 neonatal deaths were certified as due to "melaena" in England and Wales. The aetiology is not known for certain but the postnatal fall in the level of plasma prothrombin is probably the most important factor concerned.

The type of labour is irrelevant to this affection and the baby is frequently in excellent condition prior to the onset of bleeding which is generally between the twenty-fourth and seventy-second hour after delivery, though it may be delayed till as late as the eighth or tenth day, especially when umbilical in origin. Melaena is the commonest type of bleeding but haematemesia is a frequent form.

Umbilical haemorrhage is less common, while bleeding into the skin and from the nose and urinary tract is rare.

Prevention and Treatment

The treatment of haemorrhagic disease of the newborn must be prompt if the child's life is to be saved; even slight haemorrhage calls for immediate administration of vitamin K. When bleeding is severe enough to cause

obvious anaemia intravenous blood transfusion is indicated and may need to be repeated. Therefore, the need for stores of blood and for facilities for blood grouping in obstetrical practice is emphasised. The Rh. group of a female child requiring transfusion should be investigated in order to ensure that Rh. positive blood is not given to an Rh. negative girl.

There is evidence suggesting that haemorrhagic disease may be prevented when vitamin K is given by injection to the mother in labour or to the baby soon after birth (Macpherson *et al* (1940)). It is doubtful whether this is justifiable as a routine practice, for the incidence of haemorrhagic disease is not high. Nevertheless there is much to be said for giving vitamin K when labour is prolonged or complicated and when there is a definite risk of neonatal haemorrhage associated with asphyxia and birth trauma.

(b) NEONATAL ANAEMIA

The newly born baby may show an anaemia of moderate degree when suffering from any infective process, including syphilis; when birth is premature, or when the mother's diet has been faulty.

A severe degree of anaemia is more often due to loss of blood by haemorrhage than to any other cause; this is an important fact to bear in mind because blood poured out into the lumen of the alimentary tract may not, in some cases, appear at the mouth or anus for several hours after the haemorrhage has occurred, and valuable time may be lost before treatment is given. This consists in the administration of iron (crystalline ferrous sulphate) and, if need be, blood transfusion and liver extract by injection. Anaemia as a manifestation of haemolytic disease of the newborn is dealt with later.

(c) HAEMOLYTIC DISEASE OF THE FOETUS AND NEWBORN BABY

This condition which, through the work of Clifford and Hertig, Parsons, Diamond, Blackfan, Darrow, Wiener, and others is now known to include congenital oedema or dropsy of the foetus (hydrops foetalis), icterus gravis neonatorum, congenital haemolytic anaemia and probably some cases incorrectly regarded as physiological jaundice, is due to haemolysis of foetal red blood corpuscles by an antibody contained in the mother's blood (iso-immunisation). Affected subjects show increased erythropoiesis in the bone-marrow and widespread development of extra-medullary centres of blood-formation (erythroblastosis); there is frequently an increased number of immature erythrocytes in the blood stream (erythroblastaemia).

Ottenburg in 1923 was the first to postulate the theory of iso-immunisation in the pregnant woman, and in 1938 Darrow supported this idea. At about the same time Levine and Stetson (1939) happened to witness an unusual intragroup transfusion accident when a woman was delivered of a macerated foetus and developed jaundice and anuria after receiving a transfusion of her husband's blood which was believed to be of the same group (Group O). She recovered, and these workers were able to show that her serum agglutinated 80% of group O bloods, and they suggested that she had been immunised by a new antigen derived from the macerated foetus.

Landsteiner and Wiener (1940), working with the serum of rabbits which had received injections of blood from a certain species of monkey (rhesus) showed that a large proportion (about 85%) of white people have a factor in their blood cells similar to that found in rhesus monkeys and hence called the Rh. factor. Baar and Lloyd (1943), studying the fate of transfused red blood cells, demonstrated the presence of an actual haemolytic factor in

haemolytic disease of the newborn; and Levine, Katzin and Burnham (1940) showed that haemolytic disease of the newborn was the result of Rh. blood incompatibility between mother and child.

More recent studies reviewed by Cappell (1946) have shown that various subgroups of the Rh. factor exist and the subject has become one of great complexity. The essential fact, in so far as haemolytic disease of the foetus is concerned, is that the Rh. factor operates as a Mendelian dominant and that an Rh. negative woman who mates with an Rh. positive man may harbour within her uterus an Rh. positive foetus. The foetal red blood cells then contain an antigen which may stimulate the formation of specific antibodies in the mother's blood; these may pass through the placenta and cause haemolysis of the infant's erythrocytes and, in all probability, destruction of certain other cells, e.g., polygonal cells of the liver (Boorman and Dodd (1943)), and ganglion cells in the brain. If this action is intense and prolonged foetal death or a severe type of foetal disease results, namely foetal hydrops; if the action is less intense correspondingly milder degrees of the same condition may be produced.

Rarely the mother of a foetus suffering with haemolytic disease may be Rh. positive; and in a small percentage of cases the disease seems to be due to destruction of foetal cells by anti A or anti B agglutinins (Aubert *et al* (1945); Gruber *et al* (1946); Coombs *et al* (1946)). This was believed to be the cause in 4 of 48 cases reported by Boorman, Dodd and Mollison, (1944). According to Levine *et al* (1940) the A and B factors are widely distributed in the tissue fluids in 80 per cent. of subjects in A and B groups and thus inhibit the action of antibody on the corpuscles. In 20 per cent., however, ("non-secretory") they are mainly confined to the cells and then the antibody, escaping neutralisation in the fluids, is free to cause damage to the cells.

Pathological effects upon the foetus

There are variations in the blood picture; thus the degree of erythroblastæmia—usually well-marked in icterus gravis—varies from case to case, and from day to day; sometimes it is absent. Moreover the blood of a normal newborn full time infant may contain normoblasts in such numbers as to appear pathological. Again, cases of hydrops foetalis sometimes show little evidence of erythroblastæmia and in haemolytic anaemia of the newborn erythroblastæmia may not occur until the stage of recovery is reached. It should be stressed that neonatal infection and syphilis may present clinical and haematological pictures identical with those of icterus gravis.

There may be, in addition, conspicuous hepatic changes (Hawksley and Lightwood (1934); Javert (1942)) more marked in the hydropic group; and Henderson (1942) reported hepatic cirrhosis in macerated fetuses. The oedema is probably due to a decrease of blood proteins secondary to liver damage, or to capillary damage by blocking antibodies (Wiener 1946).

Clinical Types

Intrauterine death, stillbirth, premature labour and possibly abortion may all be caused by haemolytic disease; if no other explanation is obvious in any such case the possibility of blood incompatibility must be considered. The child may be born in a dropsical state (hydrops foetalis) or may show anaemia or icterus gravis. Attention should be drawn to cases which may closely resemble "physiological" jaundice, and to others in which there may be a phase of obstructive jaundice (Ross and Waugh (1936); Lightwood (1943); Skelton and Tovey (1945)).

Incidence

About 17 per cent. of the population* are Rh. negative and in one in eight marriages the wife is Rh. negative and the husband Rh. positive. However, the frequency of haemolytic disease is not nearly so high as these figures might suggest. In the large majority of cases it would appear either that there is no break in the placental barrier or that the woman does not react to the antigen by antibody formation.

Earlier estimates, such as 1 in 1,500 for icterus gravis (Hellman and Hertig (1938)) are low and suggest a failure to recognise all cases; while a recent estimate by Halperin *et al* (1945) is exceptionally high. They found that 40 out of 3,791 live born infants died from haemolytic disease and they state that there would have been more deaths if prompt treatment had not been given. Mollison, Mourant and Race (1948) state that the incidence of haemolytic disease seems to be only about 1 in 200 of all pregnancies; and the following examples from the literature may also be quoted: Javert (1942) 1 in 468; Wolfe and Neigus (1940) 1 in 568; Buxton and McDuff (1945) 1 in 516 with a foetal mortality of 57 per cent.; Cole (1947) 1 in 230 deliveries (after the 28th week).

As a rule the first child (and sometimes the second) of an incompatible couple escapes the disease or manifests it only in its mildest form. Potter and Wilson (1945) report that of over 100 affected babies born to untransfused primigravidae, none died. With each succeeding Rh. positive pregnancy the level of antibodies in the mother's blood rises in a cumulative way and the effect on the child becomes more serious. Once sensitised a woman remains so for the rest of her life. It is therefore important to recognise that the transfusion of Rh. positive blood to an Rh. negative individual at any time in life, including babyhood, can permanently immunise that individual. Thus even the first baby of an Rh. negative woman who has been transfused with Rh. positive blood may be fatally affected. Levine (1945) reports that haemolytic disease is almost twice as common among the children of Rh. negative women previously immunised by transfusion.

When the first child is affected the outlook for subsequent Rh. positive infants is very grave and Javert (1942) states that out of 27 such cases reported in the literature, only one healthy infant was born subsequently.

Prognosis

When a woman has given birth to a child suffering from haemolytic disease the outlook for the birth of a healthy infant subsequently will in general depend upon whether the father is homozygous or heterozygous; if the latter there is a 50 : 50 chance that an unaffected child will be born. The prognosis for the infant varies with the clinical manifestations. Hydrops foetalis is fatal before or shortly after birth; anaemia gravis has a fairly good outlook and the survivors do not show any permanent ill effect. Many infants suffering from icterus gravis die within a few days of birth, but some recover completely, while others develop serious complications including "kernicterus" (Schmorl (1904)); mental defects (B.M.J. Annotation (1945)) and grave damage to the central nervous system; cirrhosis of the liver and green teeth. The incidence of "kernicterus"—a condition in which there is selective yellow staining of the basal and other ganglia and the child is seriously crippled by spastic paralysis—is probably between 5 per cent. and 15 per cent. of all cases of icterus gravis. Vaughan (1946), however, reported a series of 72 cases of haemolytic disease with 20 showing serious after-effects.

* According to Lubinski *et al* (1945) the percentage is lower in the Jewish race.

The very grave outlook for cases of haemolytic disease became less serious when treatment by blood transfusions to counteract the anaemia was introduced (Diamond, Blackfan and Baty (1932); Hawksley and Lightwood (1934)); and thus, even before the discovery of the part played by the Rh. factor, the lives of many patients were saved by blood-transfusions. Colver (1938) found the prognosis to be especially grave in babies who were icteric at birth and only one out of nine of these patients survived. His "over-all" mortality was 62 per cent. and his study, which has been criticised on the ground that his series was small, suggested that perhaps one fifth or more of the survivors showed nervous sequelae such as spasticity, or mental deficiency.

ESTIMATES OF THE MORTALITY RATE IN ICTERUS GRAVIS BEFORE
USE OF RH. NEGATIVE BLOOD TRANSFUSIONS

Author	Year	Mortality per cent.
Colver (50 cases)	1938	62
Hellman & Hertig ... (20 cases)	1938	50
Wolfe & Neigus ... (20 cases)	1940	75
McKinley (37 cases)	1941	54
Javert (22 cases)	1942	54

The following figures from Birmingham (Parsons, personal communication) show the beneficial effect of Rh. negative blood transfusions:

Year	Treatment	Number of cases	Mortality per cent.
1932-1936	Serum and blood ...	42	52
1937-1941	Blood (A B O groups matched)	69	50.5
1941-May 1946 ...	Rh. negative blood ...	139	29

Treatment and Prevention

The treatment of haemolytic disease is, as yet, far from satisfactory but even with our present limited knowledge much can be done and lives can be saved (Cappell (1946)). The essential treatment of the baby born alive and suffering from any of the manifestations of this disease is to give prompt and possibly repeated transfusions with Rh. negative blood, a supply of which should always be available in obstetrical units. It should be understood that the effect of blood transfusions is simply to limit and compensate for haemolysis, though Wiener now maintains that the exsanguination—substitution technique (Wallerstein (1947)) may prevent kernicterus.

There is some difference of opinion regarding the wisdom of transfusing infants who are neither jaundiced nor obviously anaemic; but Rh. negative blood should certainly be given if the baby's haemoglobin falls to 80 per cent. or less by the second day, or to 60 per cent. at any later date; or when the erythrocytes are under 4,000,000 per 1 cmm. The volume of transfused blood needed may be calculated as follows: Body weight in lbs. $\times 40 \times H/100$, where H represents the percentage of haemoglobin necessary to bring the existing percentage to 100. In order to raise the child's haemoglobin by, say, 30 per cent., the volume of blood required will be—Body weight in lbs. $\times A \times 30/100$ ccms., where A represents the normal blood volume (usually about 40 ccs.) per lb. of body weight. Wiener states that injection of typhoid

or pertussis vaccine may prevent congenital haemolytic disease since the simultaneous injection of a stronger antigen may prevent sensitization by a weaker one (Parsons (1946)).

Expert nursing and medical care with special laboratory facilities are also required if the best results are to be obtained. If treatment is to be prompt, the diagnosis must be made early and preferably during the pregnancy. This demands routine examination of the blood of every pregnant woman with special supervision and investigation of those found to be Rh. negative and married to an Rh. positive man. When iso-immunisation is suspected foetal blood taken from the umbilical cord should be examined immediately after birth. Already in some large centres facilities are available for routine blood-grouping of expectant mothers; these facilities should be extended to cover all areas and clinics.

When the mother has had previous children who have suffered with or died of haemolytic disease, one of the main clinical objects is to achieve a live birth; in some cases the induction of labour or delivery by Caesarean section before term seems a logical treatment, but the value of such measures has not yet been established.

From the prophylactic point of view no successful treatment has yet been discovered. It is neither practical to advise an Rh. negative woman to avoid marrying an Rh. positive man, nor is it reasonable, because in most cases their children would suffer no harm; it is not yet possible to pick out the few in whom the ill effects of incompatibility would subsequently become manifest. It is, however, most important to avoid giving transfusions of Rh. positive blood to Rh. negative girls and women at any time in their lives.

Further research may ultimately solve the problem of prevention.

(d) INFECTIONS OF THE NEWLY BORN

(i) *Introduction*

Infections of the newly born and the diseases they cause constitute one of the major problems of neonatal health and hygiene. Though these infective illnesses are frequent, doctors and nurses who undertake the care of the newly born do not always recognize them and therefore fail to appreciate their significance.

Some of the peculiar difficulties and problems of the subject have been summarized by Spence (1941):

(1) The infective illnesses of the newly born may run their course with great speed. In many cases death takes place after an illness lasting less than 12 hours.

(2) Infection may occur during parturition or so quickly after delivery that deaths due to it may be ascribed erroneously to birth injury or other non-infective causes.

(3) A wide variety of diseases may be caused by one type of pathogenic organism; for example, staphylococcus aureus may cause a sudden and fatal septicaemia, an acute gastro-enteritis, neonatal osteitis, pneumonia, or "pempigus neonatorum".

(4) A pathogenic organism may give rise to an entirely different type of disease from that which it causes in an older child or an adult; for example, the dysentery organism may in the newly born cause meningitis without enteritis.

(5) Organisms that are non-pathogenic to adults may cause disease and death in the newly born.

(6) The course of an established infective disease in the newly born and the process of recovery may be entirely different from that in the older child or adult; staphylococcal osteitis of the newly born may be quoted as an example.

(ii) Incidence of Neonatal Infection

The important part played by infection in foetal and neonatal death is indicated by the figures presented in the following table; they are the findings of a pathologist (A. R. Macgregor, 1943) to a large maternity hospital where necropsies are performed on a high proportion of all live-born babies that die in hospital, and on many dead-born foetuses. The infection was not invariably considered to be the primary or direct cause of death, but in all probability it was an important contributory factor.

Age in days	Total necropsies	Number of cases showing infection	Per cent. infected
Born dead	515	31	6
1- 3	298	50	16.7
4- 7	61	29	47.2
8-14	77	55	71.4
15-21	46	34	73.9
22-28	17	14	82.4
1-28	499	182	36.5
8-28	140	103	73.6
TOTAL	1,014	213	21

These figures show that infection plays a significant part in mortality during the whole period under consideration, including foetal deaths before delivery; and that the percentage of deaths in which it is concerned, as a direct or contributory cause, increases steadily throughout the first month and is formidably high after the first week.

Figures indicating the clinical frequency of neonatal infection during the three year period 1940-42 in the Simpson Memorial Maternity Pavilion, Edinburgh have been recorded by J. L. Henderson (1943). His findings were as follows:—

Disease	No. of cases	Incidence per cent.
Conjunctivitis	1,284	15.9
Staphylococcal skin infection	550	6.8
Thrush	395	4.9
Gastro-enteritis	102	1.25
Infection of the respiratory tract	42	0.5
Pneumonia, primary	11	0.14
Pyelitis	7	0.09
Omphalitis	6	0.07
Septicaemia	5	0.06
Meningitis	3	0.04

Although much of this infective disease is not fatal, the two most prevalent conditions (conjunctivitis and skin infections) causing little risk to life, it constitutes in the mass a considerable amount of illness among young infants and most of it is preventable.

(iii) *Mode of infection*

Infection may be acquired before, during or after birth.

(a) *Antenatal infections*: Apart from syphilis, true antenatal infection of the foetus would appear to be rare. Some virus infections, e.g., measles, varicella and smallpox, can be transmitted from the mother to the foetus through the placenta. Bacterial infections, perhaps owing to the larger size of the micro-organisms concerned, are not readily transmitted in this way unless there are actual lesions in the placenta, as in the rare cases of congenital tuberculosis. Nevertheless, intrauterine infection of the foetus by gas-gangrene organisms, clostridium *Welchii* and the parasites of malaria and toxoplasmosis (Schwartzman and others (1948)) have been described. Intrauterine syphilis is a special problem and is considered on p. 69.

(b) *Intrapartum infections*: The majority of infections found in stillborn children are acquired during the process of birth. They are most common in cases of prolonged labour, especially when the membranes rupture early, for these circumstances favour the spread of bacteria from the vagina to the uterus, with consequent risk to both mother and child. The most frequent infection of this type is pneumonia (Browne (1922), Macgregor (1939)). It is caused by inhalation of infected material from the uterus or vagina, and is favoured by foetal asphyxia, which is apt to stimulate foetal respiratory efforts. Other lethal lesions due to intrapartum infection are rarely found but there are occasional cases of meningitis, pleurisy, pericarditis, etc. These are usually the result of foetal septicaemia, which may arise either from inhaled infection or from micro-organisms transmitted directly to the foetal blood stream via the placenta, which is itself likely, in such cases, to show evidence of infection. There is as yet little information concerning foetal septicaemia, but a study by Douglas and Stander (1943) suggests that it may be much more frequent than supposed and that it may be responsible for many unexplained deaths when no obvious pathological lesions are found. These workers emphasise the association of prolonged labour and instrumental delivery with such infections. Further investigations are required.

An infective lesion which causes death within the first three or four days after birth has usually been contracted intrapartum. Pneumonia is the most important example and is due, as in the type noted above, to inhalation of material in the maternal passages and may be aggravated by foetal asphyxia. Other manifestations of infection during this early period may similarly be ascribed to infection during birth, e.g., conjunctivitis, gonorrhoeal ophthalmia, oral thrush, dysentery (Greenberg, Frant and Shapiro (1940)), meningitis and other results of septicaemia. The second half of the first week is a transitional period in so far as the results of intranatal and postnatal infection are concerned.

(c) *Postnatal Infections*: From the end of the first week after birth, infection is the principal cause of neonatal death. Two infective diseases are responsible for the greater part of the mortality during this period—acute gastro-enteritis and pneumonia.

Acute gastro-enteritis sometimes occurs in epidemics in maternity hospitals and other institutions where young infants are nursed. It spreads readily in a nursery and causes serious illness and many deaths. This grave problem has received less notice than it deserves, though in recent years several reports upon it have appeared both in this country (Ormiston (1941), Cook and Marmion (1947)) and in the U.S.A. (Rice *et al* (1937)). Doubtless it is more prevalent in maternity hospitals than the number of published reports suggests. It affects infants usually after the first week; the incidence is much

higher in the premature than in the mature, and breast-fed infants are rarely attacked. The mortality rate has been as high as 70 to 80 per cent. in some outbreaks. Many efforts to discover a causative micro-organism have so far failed (Crowley *et al* (1941)). It is suggested that the infection may be a virus, or that the causative micro-organism may be one that commonly inhabits the bowel, and acquires pathogenic properties from time to time. In some cases it seems probable that the infection is parenteral.

Pneumonia is common during the later part of the neonatal period and occurs both as a primary and as a terminal event complicating other diseases; usually it manifests itself as broncho-pneumonia and it is frequently fatal. When it occurs as a terminal event, e.g., in cases of gastro-enteritis, it is often due to inhalation of vomited material and may then take a fulminating, suppurative form. This septic pneumonia due to inhalation is much more common in the newly born than in older infants and children. The bacteriology of neonatal pneumonia presents interesting and unusual features. An investigation by Macgregor (1939) showed that types of bacteria commonly responsible for pneumonia at later ages (e.g., pneumococcus, streptococcus haemolyticus, *H. influenzae*) are seldom the cause of neonatal pneumonia, which is frequently due to infection by organisms (e.g., *B. coli*, non-haemolytic streptococci) which rarely cause pneumonia in older subjects. Primary staphylococcal pneumonia, rare after the first year, is more common in the first month than at any other age. The same is true of other infective conditions, e.g., meningitis, of which the most frequent cause during the neonatal period is *bacillus coli*, an organism hardly ever responsible for the disease after the first month. These facts are important because they indicate that newly born babies are peculiarly susceptible to infection by common micro-organisms of relatively low pathogenicity; and that the results of these infections are quite different from, and much more serious than, those produced in older subjects. The signs and symptoms of neonatal pneumonia are usually obscure and atypical. Many cases are missed, or discovered only at necropsy, and many deaths due to pneumonia are wrongly ascribed to other causes.

Other forms of infection are less important factors in neonatal mortality. The risk of umbilical sepsis and the need for prophylactic precautions are well known but merit emphasis. Infecting organisms are commonly either streptococci or staphylococci; but the possibility of other types, especially pneumococci, tetanus bacilli, diphtheria bacilli and gonococci must be remembered in order that specific therapy may be employed if necessary. In some cases the umbilicus appears to be healthy; on the other hand purulent omphalitis usually causes some degree of surrounding cellulitis, and erysipelas may develop. Umbilical arteritis and phlebitis may become purulent, causing abscesses in the liver and lungs, with septicaemia, wasting and jaundice. The risk of septicaemia or pyaemia developing from other—often trivial—septic infections, e.g., staphylococcal skin lesions (Benians (1943)) must be remembered, the best safeguard being prompt recognition and treatment of the primary condition. Elliott, Gillespie and Holland (1941) have reported the probable transmission of this type of infection from the nasal passages and nasopharynx of nurses. Urinary infection (pyelitis) seldom causes death if promptly diagnosed and treated efficiently. Pneumococcal meningitis has been reviewed by Hogg and Bradley (1945). Thrush, which is liable to be endemic in maternity hospital nurseries and kindred institutions, is usually a minor disease but has its dangerous possibilities in the newly-born; severe cases are not uncommon, and in these the infection spreads from the mouth to the pharynx and oesophagus, and even to the gastro-intestinal tract (Macgregor and Henderson (1943)) and may be fatal. An outbreak in the nursery should never be regarded as unimportant, and since the infection is

readily transmitted, e.g., by nurses' hands, by oral carriers among nurses, by milk or utensils (Ludlam and Henderson (1942)) a high standard of hygienic technique is required to control it.

Tetanus neonatorum is fortunately rare. Infection generally occurs through the umbilical wound, but the organisms are not easy to demonstrate and healing of the umbilicus may appear to be quite satisfactory. In most cases symptoms occur between the second and fourteenth day after birth; the earlier the onset the more serious is the prognosis.

(iv) *Source of post-natal infection*

(a) *From the mother:* Many isolated cases of coincidental infections in mother and baby have been recorded and most obstetricians and pædiatricians have met with examples, but it is not easy to assess the importance of puerperal sepsis as a cause of infection in the baby. Figures provided by A. C. Bell (1943) from Queen Charlotte's Maternity Hospital and its Isolation Block have some bearing on this problem. During the years 1934 to 1938 post mortem examinations were carried out on 201 babies dying in the neonatal period and the following table shows the findings as regards neonatal infection:—

Hospital	No. of cases	General Infection (excluding pneumonia)	Total Infants
Queen Charlotte's			
(a) Main Hospital ...	186	9 (5%)	31 (16.7%)
(b) Isolation Block ...	15	7 (47%)	10 (66.6%)

(The baby was often admitted with the mother to the Isolation Block, though in later years this practice was discouraged owing to the risk to the baby.)

These figures are small, but the percentage differences would appear to be significant and they support the claim that puerperal infection in the mother may cause fatal generalized infection in the newborn. As regards pneumonia there is little difference between the two groups and Bell suggests that this is because pneumonia resulted from intranatal infection. The details of the cases of generalised infection have special significance. In the Isolation Block, 4 of the 7 babies were infected with haemolytic streptococci and so were their mothers. In a fifth case both mother and baby had a *B. coli* infection. In Queen Charlotte's Main Hospital however, none of the neonatal deaths was due to haemolytic streptococcal infection, and in only one did the mother have an infection with organisms similar to those which attacked the child (pneumonia).

It should be emphasised that the above figures are derived from a hospital whose staff have paid considerable attention to the problem of preventing infection, both in mother and child.

(b) *By feeding:* It has been frequently observed that a majority of the babies who develop gastro-enteritis are bottle fed and that premature babies are especially susceptible. Though the responsible organisms have not been isolated the evidence strongly suggests that infection is by way of contaminated milk or unclean bottles or teats. Thus the infection may be spread directly from baby to baby, or indirectly when the hands of the nurse, wash-basins, etc., become contaminated.

Although breast milk is an infrequent cause of neonatal infection, it must be recognised that pathogenic organisms sometimes reach the baby by this

route, especially if the breast tissue is infected (mastitis); in such circumstances breast feeding should be suspended at once. Haemolytic streptococci have been demonstrated in breast milk and staphylococcus aureus is frequently found, but the strain is usually non-pathogenic.

(c) *By other contacts:* It is becoming increasingly recognised that infection with staphylococcus aureus is a common cause of neonatal morbidity and mortality. According to Knott and Blaikley (1944) the pathogenic organisms are nearly always group A or B, and they are carried into maternity wards by attendants (medical and nursing), visitors or mothers. They are to be found in dust and bedclothes, as well as upon human carriers and they are most difficult to eradicate. Outbreaks of minor infection, such as mastitis in the mother or septic rashes in the babies and sometimes more serious conditions such as pneumonia and pemphigus neonatorum may be caused. Direct transfer from patient to baby and from clothes or ward dust to baby has been demonstrated. Moreover it should be remembered that the respiratory tract and hands of nurses may also become infected from the babies, and so the organisms may be spread from one infant to another. The fact that strict attention to the possibilities of such modes of transfer may lead to a virtual disappearance of staphylococcal infection (Knott and Blaikley (1944)) is additional proof that this risk exists in maternity units.

(v) *Neonatal Infection in Institutions*

The advantages and disadvantages of institutional midwifery, as opposed to domiciliary midwifery, have long been argued and general agreement has not been reached. But it is certain that an ever increasing number of women desire their confinements to be in hospitals or nursing homes, and in some urban areas 70 per cent. of all labours take place in institutions. It is important, therefore, to emphasize that neonatal infection is much more liable to occur in hospital than in domiciliary practice, and special care is necessary if it is to be prevented. The chief reasons for this prevalence of infection amongst babies born in hospitals are:—

1. Cross-infection was imperfectly understood and its importance insufficiently appreciated when most of the present maternity hospitals were planned. They were built on the mistaken principle of aggregating the infants in small nurseries to economise space, labour and expense. A barrier-nursing technique is difficult, if not impossible, to maintain in such conditions; and the danger of dust infection is considerable.

2. The general standard of knowledge in the medical and nursing professions about the care of the newborn baby is unsatisfactory. There is a failure to appreciate a baby's susceptibility to infection and the serious consequences that may arise.

3. *Nursing staff arrangements.* (a) The number of nurses is inadequate for the maintenance of a high standard of infant care, especially in an unsuitable building.

(b) Much of the infant nursing is in the hands of pupil midwives who may be certified (general trained) nurses though without any experience of infants. Only a few of the pupil midwives have had the advantage of experience in infant nursing by taking a sick children's or fever nurse's training.

(c) Pupil midwives spend only a few weeks of their training in nursery duties because they must gain experience in all the various departments of the hospital and the hospital district; as a result, much of the nursery work is done by beginners.

(d) The quality of the infant nursing suffers not only from inexperience but also because of the heavy demands made on the pupil midwife's time and energy by lectures, tutorials and study which are necessary for her to pass the examinations of the Central Midwives Board.

(e) Only a very small proportion of pupil midwives take up midwifery as a career; the majority have no intention of doing so, and they may, therefore, have little sustained interest in the technique of nursing infants.

(f) Nurses who feel ill may not be encouraged to report the fact. It is dangerous to infants when nurses continue on duty while suffering with minor infections, particularly naso-pharyngitis, diarrhoea and sepsis of the hands; but the administrative staff tend to ignore this hazard because they have many staffing difficulties to meet.

4. *Nursing Technique.* The common changing room and changing table and the communal use of weighing scales, baths, ointment pots and thermometers all facilitate cross-infection. Door handles and tap handles transmit infection from the hands of one nurse to another. Transport trolleys to convey infants from nurseries to wards for feeding invite cross-infection, because babies are often closely packed upon them; this is a labour-saving device necessitated by inadequate staff. Nurses rarely wash their hands after attending to each child and in some hospitals they do not wear gowns as a routine. Artificial feeds are usually prepared by nurses who also change the infants' napkins. The disposal of soiled napkins is often unsatisfactory, and ill health, e.g. septic fingers, among the staff of the laundry may result in infection of babies by the clothes and napkins.

5. Routine bacteriological examination of the staff and ward maids in maternity units is seldom practised.

6. Routine bacteriological examination of all mothers on admission is not practised.

7. Visitors are not adequately restricted or controlled.

8. Isolation facilities for ill babies are usually insufficient and may be non-existent. Cross infection is very common in nurseries intended for sick infants because of overcrowding and lack of barrier nursing.

General Conclusions regarding dangers of infection to newborn babies.

1. Though infection plays a relatively small part in causing foetal mortality, it is an important factor in neonatal mortality and morbidity.

2. A majority of neonatal deaths occurring after the first week are due to infection of one type or another; these deaths should be regarded as preventable. Effective control of infection offers one of the most hopeful means of reducing neonatal mortality and morbidity.

3. Premature and immature babies are especially susceptible to infection.

4. Pathogenic micro-organisms may be transferred directly from an infected mother to her infant.

5. Infection with the common bacterial agents e.g. staphylococci, *B. coli* etc., occurs by the usual channels including droplet infection, and contact with infected hands, utensils, clothing etc.

6. Unless special precautions are taken, institutional midwifery carries considerable risk of infection to newly born babies. Recommendations to reduce this risk are:

(a) Each baby should have its own equipment and toilet requisites such as thermometers, ointment pots, etc. The weighing machine is an appliance in common use and precautions can be taken to prevent it becoming a means for transfer of infection. Clothing should be sterilised whenever possible.

(b) Each baby should have a separate bath; or babies should not be bathed while in hospital. Frequent bathing may damage the baby's skin and predisposes to skin infection; there is much to be said for limiting the number of baths to two or three in the first fourteen days of life.

(c) The common "changing" room and transport trolleys should be abolished. Babies should be "changed" in or beside their own cots. Soiled napkins should be dropped into a special container and should not be touched thereafter by any nurse. Destructible napkins should be used whenever possible. The floors of nurseries should be treated to reduce the risk of dust infection.

(d) There should be a nurse's and doctor's gown per cot. All in attendance should wear masks and should be proficient in mask drill.

(e) The nurse should wash her hands before and after attending to each baby, and after removing the gown. Nurses should be provided with rubber gloves, especially during winter.

(f) All doors should be swing doors without handles; taps should be of the foot or lever type to avoid the use of hands.

(g) Routine throat swabbing of the staff and ward maids would reveal carriers of pathogenic organisms, such as coagulase-positive staphylococcus aureus.

(h) Routine bacteriological examination of all mothers on admission would be desirable; any showing virulent staphylococcus aureus or other pathogenic organisms should be isolated until clear of these organisms.

(i) Visitors, who are liable to introduce infection, should be strongly discouraged; husbands only should be allowed admittance, except in special circumstances.

(j) Infants who show any infective condition, however slight, or who are suffering from any undiagnosed condition, should be isolated immediately.

(k) The desirability or otherwise of separating mother and infant when either has an infective or undiagnosed illness depends on individual circumstances. Separation is not always necessary, particularly when it is the baby who is infected. Mothers of infants with superficial staphylococcal infections run an increased risk of mastitis, but this is a small risk and may be ignored. On the other hand, the development of frank infection in the mother may be an indication for removing the baby from her. Babies should not be admitted with their mothers to "puerperal sepsis" blocks.

(l) Propaganda about infection and its dangers should be intensified and maintained. An "infection conscious" outlook should be fostered by lecturers, sisters and staff nurses, and the junior nurses should be given lecture demonstrations by the clinical and laboratory medical staff.

SYPHILIS

In general the placenta protects the foetus in utero from maternal infections. The most important exception is syphilis and the spirochaeta pallida

readily infects the placenta and passes to the foetus. This seldom occurs, however, before the 20th week of pregnancy and it is now generally recognised that syphilis does not cause early abortions.

(1) *Incidence.* This seems to vary considerably in different parts of the British Isles. Thus in University College Hospital during the seven year period 1932 to 1938 inclusive, routine Wassermann tests upon expectant mothers showed that 126 (1.13 per cent.) of 11,131 women were syphilitic (Browne (1943)). In Glasgow, Cruickshank (1924) found the incidence to be between 9 per cent. and 10 per cent., and in Edinburgh, Gladys H. Dodds (1927) found that the incidence in 1,000 women was 5 per cent. In Edinburgh, however, there were beds reserved in the antenatal department for the treatment of syphilitic expectant mothers and this doubtless resulted in syphilitic women being specially referred. In University College Hospital this special provision is not made and it is probable that the incidence found there is fairly representative of urban districts generally in this country. Furthermore it must be remembered that a large proportion of syphilitic expectant mothers receive more or less adequate treatment during pregnancy and therefore many of their infants are born free from syphilis, and the baby of an untreated syphilitic woman is not always syphilitic. On the other hand there is no doubt that a woman with a negative Wassermann reaction but with latent syphilis sometimes gives birth to a syphilitic infant. After making allowances for all these difficulties, and bearing in mind that routine Wassermann tests are by no means universal in ante-natal clinics throughout the country, it seems probable that the foetus is infected by syphilis in about 1 per cent. of all pregnancies. Many of these infected infants are not born alive. If we assume that one half are born alive (a low estimate, for Cruickshank (1924) found that 83 viable infants were born alive of 94 women with positive Wassermann reactions) we arrive at the conclusion that about 3,000 congenital syphilitic infants are born in Great Britain every year. Many of these are premature—20 per cent. in Cruickshank's series.

Fildes' (1915) estimate coincides closely with this. Four cases of congenital syphilis were found among the offspring of 677 married women in East London belonging to the "respectable labouring classes" (0.59 per cent.).

The frequency of congenital syphilis as a cause of neonatal death is difficult to estimate. Among 153 consecutive cases of neonatal death Browne (1943) found only four in which the cause of death was undoubtedly syphilis, proved by the presence of spirochaetes in the foetal tissues. This is an under estimate, however, as the spirochaete cannot always be demonstrated in fresh syphilitic infants; and one of the infants in which spirochaetes were not found was the twin of a macerated foetus in whose organs they were specially numerous.

During the recent war the incidence of syphilis in this country has increased, and it might be expected that this will result in a rise in the incidence of congenital syphilis during the next few years. So far, however, there has been no indication of this, and it would appear that the use of more efficient methods of treatment has more than compensated for the increased incidence of syphilis in adults. Inherited infection, therefore, is not at present a common cause of stillbirth and neonatal death.

Third generation syphilis. This is always difficult to prove as it is seldom possible to exclude reinfection of the mother, especially when she admits the possibility of reinfection. Theoretically there is no reason why third generation syphilis should not occur; for an untreated congenitally syphilitic

child harbours spirochaetes which, persisting to adult life, might be transmitted to the third generation just as in acquired syphilis. Many convincing cases are recorded in the literature; Nabarro (1933), for example, cites 17 families.

Prevention and Treatment. The ideal is clearly that adults should avoid infection by syphilis; this can be achieved only by clean living and it is largely a social and educational problem. In any case a considerable time may elapse before syphilis is eradicated and in the meantime all possible steps should be taken to protect the unborn child of the syphilitic mother. The first essential is diagnosis early in pregnancy, and to secure this it is agreed that all pregnant women should have serological tests carried out as a routine; but there is need for care in the interpretation of the results. When the diagnosis is made sufficiently early in pregnancy and the treatment is in the hands of an expert, there is every reason to expect that the baby will be born healthy; but as an additional precaution, the child may need further treatment for some time. An infected woman requires special supervision during her subsequent pregnancies.

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X. ADDITIONAL GENERAL RECOMMENDATIONS

The following observations and recommendations of a general nature are *additional* to those with which the various preceding chapters have concluded. They are based on the evidence contained in this Report and relate directly to the prevention of stillbirth, neonatal death and disease. Portions of a Report (1944) prepared by the Royal College of Obstetricians and Gynaecologists on a National Maternity Service have been included, together with abstracts from Reports prepared by the British Paediatric Association.

Some of the recommendations in the body of the Report are again emphasized in this general summary.

(1) **SOCIAL AND ECONOMIC CONSIDERATIONS—DIET.** Every family should be assured an income sufficient to provide all its members with an adequate diet. Education of the public in wise spending, cooking and the need for balancing the diet should be encouraged. The facilities whereby a pregnant women can obtain an extra supply of milk, vitamins, iron and other dietary supplements should be provided in peace as in war. Lest other members of the family should benefit at the expense of the mother, one well balanced meal a day in a Restaurant or Canteen might be provided for all pregnant mothers. Many attempts have been made to define the standard energy requirements during pregnancy and lactation but no unanimity has been reached. It is, however, permissible to suggest tentatively that 2,500 calories per day during pregnancy and 3,000 during lactation represent the average needs. The wide variations in individual requirements—both as regards total food intake and particular items such as minerals and vitamins—should be appreciated and every effort should be made to supply these needs by natural foodstuffs rather than by vitamin concentrates and other supplements.

Well planned feeding, however, is not enough; adequate housing accommodation, open spaces in the cities, facilities for recreation—everything that goes to make a healthy life is required. Moreover it must be realised that the full effect of these benefits will not be manifest until the babies of today, born and bred in healthy circumstances, reach parenthood in their turn. Again, the provision of proper living conditions for all sections of the community will be insufficient unless the people are educated to make full use of them.

(2) **THE FAMILY UNIT.** We would associate ourselves with all those in the Churches, in public life and amongst social workers of all creeds, who insist that the moral and spiritual fibre of the community is closely bound

up with a high standard of family life. All are aware that the infusing of higher moral and spiritual values into the families of a country has a profound bearing on physical and mental health. For example, we are convinced that the main attack upon the recent increase of venereal disease should be a raising of the standard of sexual morality and fidelity in marriage, a standard which was lowered during the war years as a result of the upheaval in family life.

We are specially concerned with those social and economic influences that bear upon the security of the family unit, particularly in relation to the health of the mother and child. We would plead that early marriage be encouraged and we believe that the extension of measures for social security is an important means to this end and that it has a sound biological foundation. Apart from the advantage of early marriage in relation to sexual morality and its possible bearing upon population problems, it has been shown that earlier marriage has a direct bearing on the health of the mother and child. Whilst it is true that the risks associated with childbirth at a later age may be minimised by skilful care and need never be a deterrent to marriage at any age, it must be accepted that in large groups these risks attain statistical importance and have a bearing upon the stillbirth and neonatal death rates. Other factors of considerable importance for family health are the size and spacing of the family. It should be one of the functions of the Maternity and Child Welfare Service to make available to the public the best possible advice on these and kindred subjects.

It need hardly be emphasised that a mother's first duty and obligation is to the home; hers is undoubtedly the most important influence in the family circle. The health—both physical and psychological—of the young child depends upon having reliable and loving parents to whom it can turn with confidence and trust. This relationship, in so far as the mother is concerned, begins ideally with successful lactation. During a period which at a minimum extends over the last three months of pregnancy and the first six months after parturition, the mother should be free from all responsibilities outside the home which might interfere with pregnancy and lactation.

The Social Insurance Act contains provisions for the pregnant woman who ordinarily takes employment outside the home. In so far as they go, these provisions are to be welcomed; but steps should be taken to see that the monetary benefits are put to the use for which they are intended. This is not possible unless and until home helps and domestic workers are available in sufficient numbers.

The wider aspect of the place of women in society must be considered briefly. It is obvious that, if girls receive good education and a training which will enable them to bring up their children according to the best standards, they must also be given good living conditions and the assistance of labour-saving devices. The mother will not be able to undertake the care of a family of four young children without, say, the help of such communal services as home helps and nursery schools.

The home help has long been recognised as an important assistant in a complete maternity and infant health service, but local authorities have been unable in the past to make proper use of the statutory powers they possess in this respect. Whilst a young mother is lying-in at home or in hospital or is laid aside with illness, the home help deputises for her, and this assistance may be invaluable. In our country, the women engaged in this service have seldom received special training in minor nursing and the feeding and management of infants, as is required in some countries, such as Holland.

We are of the opinion that if the home helps were given a definite status with prescribed training and adequate remuneration, they would contribute in a material way to maternal and infant health. For these reasons we give our warmest support to those who advocate a wide extension of the home-help service.

The Education Act of 1944 provides for Nursery Schools suitable for children over two years of age. We believe such schools to be of great value—not only for the part they play in fostering child health directly but because they relieve the over-worked mother and are thus directly beneficial to children.

During the war there was a great expansion of Day Nurseries in order that women might take part in industry; there will always remain a place for day and residential nurseries for special occasions, for instance when there is illness in the family and when the mother is lying-in. In general, however, as we have indicated above, these family emergencies are better dealt with by home helps.

EDUCATION IN SEX AND IN PARENTHOOD.—A sound knowledge of the laws of health must be an essential part of the equipment for parenthood in both sexes; and education for healthy living should include sex education. Health education begins in the good home or nursery school with the inculcation of good habits. The child's questions about the functions of the body, including reproduction, should be answered simply and truthfully, and the child should have acquired some knowledge of anatomy and physiology, including reproduction, before the age of puberty is reached. It is important that this knowledge should be gained as part of general health education, before sex has become an individual emotional urge. On this factual foundation the adolescent learns the code of conduct which should govern the relationship between the sexes and comes to appreciate the proper use of the sexual function in respect both of the individual and the community. The stability of family life is likely to suffer when there is no clear understanding of the higher or moral aspects of sex. Special instruction in more detailed aspects of sex physiology will be necessary before marriage.

Generally speaking this teaching to young people has been avoided in the past and it is high time that the many taboos which have pervaded the subject of sex should be dispelled. At present we face the difficult task of educating adolescents who have acquired their knowledge of sex mainly in undesirable ways and who possess little real knowledge of healthy living.

Mothercraft teaching in schools and the training given to girls in nurseries and nursery schools is excellent, but care must be taken lest too narrow an outlook is given to young women; the education should be directed to inculcating general principles and attitudes rather than details of methods of cooking and infant care—at least while the girl is at school.

The question of training boys and young men in the duties of parenthood must also receive consideration, and in this connection we quote the findings of the Swedish Commission of Population:

“If it be considered desirable as a matter of principle, which the Commission feels is the case, to strengthen the position of the family in the minds of youth and to influence their attitude towards the family in a positive direction, then the Commission feels that boys and young men should not be excepted. Ever since the beginning of the industrial revolution, the man and the father have tended to be separated from family life. This is one of the most disruptive forces in the development of the family. The Commission feels that in the modern family, and especially in the modern family in which the wife is gainfully employed, at least in the early years

of marriage, the man's participation in household duties is necessary for co-operative and harmonious living. Distorted attitudes and habits exist towards this in all social classes. These constitute a danger to the family as an integrated joyous co-operation."

DOMICILIARY AND INSTITUTIONAL MIDWIFERY.—There has been much controversy about the relative advantages to both mother and child of parturition in the home or in hospital. It is said that the home is the natural place for the baby's birth and that its atmosphere has a psychological effect on the mother, tending to promote easier labour and successful breast feeding. The mother receives individual attention, and minor difficulties with breast feeding can be more easily overcome. Nevertheless, the mother at home is seldom free from her anxieties, and even when she is lying-in she tends to resume her domestic responsibilities. Moreover, under present conditions few mothers can afford to employ whole-time nurses even if these were available in adequate numbers. The majority of mothers delivered at home have, therefore, to be content with a short visit from the district midwife twice daily, and for the most part the newly born baby is left to the care of the mother and her relatives; there is in fact less nursing and medical supervision in the home than can be provided in hospital. Few houses have sufficient space to enable them to provide proper facilities for the conduct of labour. Finally, should any abnormality arise during pregnancy, labour or puerperium endangering either mother or child, the necessary treatment can seldom be given except in hospital.

On the other hand the risk of infection in the home to both mother and child is small, provided that reasonable precautions are taken against the spread of the common cold and other contagious disorders.

It may be claimed that domiciliary midwifery gives a high degree of safety to both mother and child so long as two conditions are observed:—

(a) First class antenatal supervision, including the careful selection of cases suitable for delivery at home; this selection must take into account the interests of the baby as well as those of the mother, but in practice these interests are seldom antagonistic. All primigravidae, all women who have had four children or more, and all whose labours are likely to be abnormal in any way, as well as those whose home conditions are definitely unsuitable, should be delivered in hospital. This rule should also apply when the baby is expected to be small or weakly; thus the onset of premature labour should be regarded as an indication for admission to a fully equipped hospital. A multiple pregnancy should be dealt with in hospital.

(b) Domiciliary midwifery should be conducted in close liaison with a maternity hospital; and the staff, both obstetrical and paediatric, should be available for attendance in the patient's home when necessary. It should be possible to transfer to hospital, when necessary, any sick or weakly baby and the mother should be transferred with the child. There is at present a great need both in maternity hospitals and children's hospitals for accommodation suitable to receive both mother and child after delivery.

In favour of institutional midwifery it must be accepted that if emergency admissions are excluded the results for both mother and child in well conducted hospitals are better than those for the country as a whole. Moreover, the number of women seeking admission to hospital for delivery is steadily increasing, and although in 1944 there were already available in England and Wales sufficient maternity beds for 50 per cent. of the mothers, the accommodation is full to capacity; indeed, there is serious risk, both to

mothers and babies, from overcrowding. In some areas, for instance in London, the percentage of babies born in hospital is at least 70 and may well become higher.

The chief risk to be avoided in hospital midwifery is the spread of infection, not only among the mothers, but more particularly among their babies; institutional midwifery can be made remarkably safe for mother and child but it should be stressed that this can be true only when the standard of medical and nursing care, and of administration, is of the highest order. There is need for great and sustained effort on the part of all concerned if the risk of infection is to be reduced to a minimum. It might be desirable to return many of the mothers and babies to their homes a few days after delivery in order to safeguard the child from the risks of contagion, but this would seldom be in the interests of the mother; thus it becomes increasingly important to adopt all possible methods of preventing cross infection in hospital nurseries.

The period of pregnancy and "lying-in" offers great opportunities for instructing a woman not only on matters pertaining to her own health but on the care of the baby, its clothing, feeding and general management. Education of this kind should be an important feature of antenatal and postnatal care and the medical and nursing attendants should demonstrate the proper management of babies to mothers during the lying-in period. The accommodation of mothers and babies together as recommended below is conducive to this.

Private Nursing Home Midwifery

Nursing Home practice is rapidly decreasing and little reference need be made to it here. We believe that midwifery in nursing homes which are not well equipped and which are not restricted to maternity work brings added danger to the baby and to the mother. Nursing homes that admit surgical and medical cases as well as maternity cases have the disadvantages of both hospital and domiciliary midwifery and the advantages of neither.

THE PLANNING AND CONDUCT OF MATERNITY HOSPITALS

Maternity hospitals should be planned and conducted in such a way as to safeguard the health of mothers and their babies and to ensure as far as possible that every baby on discharge from the hospital is breast fed and in the care of a mother who has been instructed in the management of her child.

Whenever practicable maternity units should be closely linked with or form part of a general hospital; close co-operation with the paediatric and gynaecological departments is particularly necessary. The details of accommodation will vary with local conditions and it is impossible to lay down hard and fast rules. Some maternity hospitals admit a high proportion of abnormal cases and in these there will be a correspondingly greater need for arrangements for the treatment of premature and sick babies. In all cases it is necessary to ensure maximum dispersal of patients and the prevailing system of accommodating mothers in wards and of crowding their babies into small nurseries is dangerous from the standpoint of infection and must be strongly condemned.

Ideally each mother and her infant should have a separate room equipped with wash-hand basin, baby bath, and every requisite for both mother and child, including a separate thermometer for each child. Mothers of the hospital class, however, usually object to isolation from their fellows, and for them the best arrangement is probably a compromise between single rooms and large wards, i.e., wards of four to six beds only, each accommodating the mothers

and their babies. These small wards should be fully equipped for the care of both mother and baby and the infant should be bathed and changed in the ward within sight of the mother. In the opinion of some authorities, these wards should be divided into cubicles by transparent partitions, each cubicle being separately equipped for the treatment of mother and baby. If, however, a system of barrier nursing is conscientiously practised, it might not be necessary to carry out such strict isolation. Until hospitals are replanned it seems probable that it will be necessary to continue the use of nurseries; but the number of babies occupying them should be strictly limited and partitioning would be advantageous.

Nursery accommodation and isolation facilities for sick babies in hospital practice

There is some difference of opinion regarding the disposal of sick newly born babies; some hold the view that they should be transferred to a childrens' hospital, which means that the mother should be transferred with the child. Perhaps the best solution of this problem would be to provide a separate block for the care of sick babies. If it be decided that the accommodation for sick babies should be within the maternity unit, it will be necessary to provide isolation cubicles for the infant, or single rooms in which both mother and child can be placed. A hospital with 100 lying-in beds would probably need accommodation for 10 to 15 sick infants and this would include provision for those babies suffering from some form of infection requiring isolation. When the baby is not with its mother in a single room it should be isolated in one of a series of cubicles with glass doors opening on to a corridor, and corridor and cubicles should be air conditioned. There should be a wash-hand basin, together with other necessary equipment in each cubicle and arrangements should be made to darken the cubicle when necessary. Piped oxygen is an advantage.

Sick infants should have a special staff (day and night) of nurses trained as sick children's nurses, who should not take part in the other work of the hospital. Pupil midwives should receive instruction in the care of sick babies.

Recommendations for immature and premature babies

In addition to the special nursery accommodation mentioned above it is necessary to have cubicles for premature infants and the number will depend upon the type of obstetrical work done. In maternity hospitals that deal with a relatively high proportion of abnormal cases, 10 per cent. to 20 per cent. of the infants may be immature; in hospitals that accept a higher proportion of normal cases, the prematures will number only 5 to 10 per cent. of the infant population. In the cubicles for immature babies the air should be conditioned as to temperature and humidity, and some of the cots should be of special design; each cubicle should be separately and fully equipped for the care of the baby. The nursing staff must be adequate in number both by day and night; the senior nurse should be specially trained in the care of premature babies and should not be engaged in other duties. Pupil midwives should receive instruction in the nursing of premature babies.

Medical Staff required for Hospital Midwifery

(a) *Obstetricians.* The general plan of arrangements which already obtains in the larger teaching centres, i.e., visiting consultants, senior resident obstetricians and junior resident obstetricians should be extended to cover all institutions practising midwifery.

(b) *Anaesthetists.* In view of the growing importance of anaesthesia and analgesia in obstetrics and their significance in relationship to both mother

and child, all the larger maternity hospitals should employ a resident anaesthetist. The number of labours conducted in the smaller hospitals might be insufficient to justify this; in that case one of the resident obstetricians should have received special training in obstetric anaesthesia.

(c) *Paediatricians*. All newly born babies (healthy, premature and sick) in a maternity hospital should be in general charge of a paediatrician who is experienced in the work; he should be a "full" member of the staff and should advise on the general paediatric policy of the hospital in the closest co-operation with the obstetricians and with the public health medical officers of the district. The assistance required by the paediatrician will naturally depend upon local conditions, such as the size of the hospital; the minimum would be one assistant paediatrician (undergoing training in neonatal work), together with the services of one or more resident medical officers who should have had previous experience in paediatrics and should be competent to carry out special treatment, such as intravenous infusions.

(d) *Bacteriologists*. The control of infection requires that bacteriologists should be invited to take an active part in studying the conditions under which the work of the hospital is conducted, in the theatres, the wards and the nurseries.

In teaching centres all the above-mentioned specialists should be members of the departments of obstetrics, child health, anaesthesia and bacteriology in the University or Medical School.

The closest co-ordination should be secured and full advantage should be taken of the facilities for research and for the teaching of undergraduate and postgraduate students, nurses, health visitors and mothers.

Nursing Staff in Maternity Institutions

A maternity hospital requires a very large nursing staff because each lying-in bed represents two patients—mother and baby—and because of the special precautions necessary to prevent infection both in babies and mothers, the special care required for proper feeding of babies and the instruction of the mother in breast feeding and child management. Allowing for off-duty times it can be reckoned that the lying-in wards need as many nurses as they have beds (i.e., one nurse for each mother and child—though not all will be on duty at the same time). Furthermore an adequate number of ward maids and orderlies is required to relieve the nurses of all except strictly nursing duties. There must be a sufficient number of day and night nurses properly trained in the care of the newly-born and able to impart their knowledge to trainees. Sisters and Staff Nurses should be on the Sick Children's Nurses' Register and should hold the certificate of the Central Midwives' Board.

The health of the nurses and ward orderlies must be supervised and any nurse who develops an infection such as a head cold, sore throat, diarrhoea, septic finger or the like should not be allowed on duty until she is declared free of infection by the physician in charge.

The Central Midwives' Board should include at least one paediatrician and paediatricians might be asked to take part in its examinations.

Arrangements for Domiciliary Midwifery

Throughout this report the term "obstetricians" has been used to denote all those who practise midwifery; it includes general practitioners as well as specialists. The general practitioners together with midwives are mainly responsible for the domiciliary work. If the best results are to be obtained doctors should have had postgraduate training as residents in maternity hospitals and should there learn not only the care of the mother

but also of the newborn child. Moreover they should maintain a constant liaison with the maternity hospital in their area; and the staff of the hospital, both obstetricians and paediatricians, should be readily available to assist the general practitioner in the patient's own home when necessary. The separation, for administrative purposes, of domiciliary and institutional midwifery which is one of the provisions of the National Health Service Act should not be allowed to interfere with the close linking of the work in the two spheres. Domiciliary midwifery can only be made really safe for mother and child when it is based on and supported by all the facilities of a nearby hospital.

Auxiliary Services

The application of recent knowledge and modern methods to the management of the newly-born demands certain other facilities which are not yet available in many hospitals in this country.

(a) *Breast Feeding Clinics*: It frequently happens that mothers who successfully breast feed in hospital fail to do so as soon as they return home at the end of 10 to 14 days. Difficulties encountered at that time are often easy to surmount if expert advice and encouragement are available. Much can be done in such cases by health visitors, but an additional suggestion is the institution of a special "breast feeding clinic," in the charge of an experienced staff, medical and nursing, where mothers can attend and stay for several hours in comfort, having two or three successive feeds supervised. Any mother having difficulty with breast feeding at the time of her discharge from hospital might be specially referred to such a clinic.

(b) *Breast Milk Bank*: Hospitals should set up a human milk bank where breast milk can be stored for the use of babies unable to receive milk from their mothers. It is possible that these banks would also be able to supply milk for necessitous babies outside the hospital.

It has been proved in some centres that one or two enthusiastic sisters, provided with a car to enable them to visit the homes of nursing mothers, can obtain useful supplies of breast milk which can then be made available for infants who would otherwise be weaned.

(c) *Feed Preparation Room*: Each hospital should have its own central milk kitchen specially reserved for the preparation of artificial feeds and for the instruction of nurses in such methods. It should have its own staff and none of the feeds should be prepared by nurses engaged in ward work.

(d) *Baby Clothing*: Many maternity hospitals find difficulty in maintaining an adequate laundry service for napkins and baby clothes. Special care should be taken to ensure an ever ready and ample supply of such articles which should be washed and sterilised before being used. Whenever possible destructible napkins should be used.

(e) *Blood Banks*: The institution of blood banks as a war-time measure has resulted in the saving of lives both of mothers and babies. It is imperative that blood banks should be continued and that maternity hospitals shall have a ready supply of all types of blood, particularly Rh. negative blood for babies suffering from haemolytic disease.

INFANT CLINICS

Infant welfare clinics cater mainly for babies after the newly-born period but they are valuable in the study of neonatal disease, birth injury, etc., and the usefulness of different methods of treatment. One or more infant welfare clinics in the personal charge of paediatricians should be held in the hospital for the benefit of both babies and mothers who should be instructed to report

periodically after their discharge if they live in the vicinity of the hospital. A number of social workers and health visitors should serve on the staff of these clinics and should visit the patients in their homes to ensure adequate supervision. Mothers and their babies from outlying districts should attend a clinic nearer to their homes. Particular attention should be paid to infants that are born prematurely and there is much to be said for setting up special clinics for the follow-up study of certain types of neonatal ill health.

ANTENATAL CARE

Although antenatal clinics have been in operation for some thirty years it is well known that the results of antenatal care have not fulfilled expectations. This is discussed at some length in the Report on a National Maternity Service prepared by the Royal College of Obstetricians and Gynaecologists in 1944 and it is pointed out that some of the reasons are:

(1) Many clinics are staffed by whole-time members of the public health service who take no part in the conduct of the confinement and are out of touch with obstetric practice proper.

(2) The number of antenatal beds in hospitals is usually inadequate.

(3) The clinics are too crowded.

(4) It must be recognised that antenatal care has its limitations; it cannot compensate for poor care during delivery. Unforeseen complications are liable to arise during labour, even when antenatal care has been expert.

In the conduct of antenatal care it is suggested that:—(a) The life and health of the baby should be kept in mind as well as the interests of the mother; this must include supervision of the mother with a view to successful lactation. (b) All clinics should be staffed by doctors actively engaged in obstetrics; they should be responsible for the delivery of the patients attending the clinic. (c) Two types of clinic should be recognised—(i) consultative—situated in hospitals, and staffed by obstetricians of considerable experience. To these would be referred patients for whom special obstetrical advice is needed. (ii) Educational and supervisory.—In these clinics the mother's health should be kept under routine supervision, but more attention should be paid to her education. The mother should be instructed in the hygiene of pregnancy, and should be prepared psychologically for labour and motherhood. She should be taught cooking and infant feeding and management. Lectures and demonstrations by health visitors and nurses could be arranged. (d) Greater care should be paid to those conditions which may affect the life and health of the foetus, e.g. general ill health of the mother, venereal disease, etc. Serological tests for syphilis should be carried out routinely on all pregnant women and it is recommended that a study of the blood groups should be made in each case so that Rh. incompatibility may be recognised as soon as possible.

CARE DURING DELIVERY

The prevention of neonatal death and illness is largely in the hands of the obstetrician and his aim should be to hand over to the mother and paediatrician a living, mature and healthy child. The obstetric tradition is to consider the interests of the mother first; but recent advances in obstetrical technique have reduced in number the occasions on which the claims of mother and child are in conflict. It should be more widely recognised that the treatment which is best suited to the needs of the mother is in most cases also best for the child, and vice versa. The maternal mortality rate has been reduced to such a low figure that the time has come when obstetricians can properly pay more regard to the outlook for the child when deciding upon

treatment, e.g. in cases of toxæmia of late pregnancy, prolonged labour due to inefficient uterine action and placenta prævia. It seems probable also that the indications for episiotomy, forceps delivery and for Caesarean section should be considered afresh in the interests of the child.

EDUCATION OF DOCTORS, STUDENTS AND NURSES

It is abundantly clear that steps should be taken to stress the importance of the life and health of the newly born baby. The teaching of medical students, doctors and nurses needs to be revised and amended in order to ensure that this is achieved.

RESEARCH

In the earlier part of this report it was made clear that in many respects our knowledge of the causes of neonatal death is incomplete and the prevention and treatment of some forms of neonatal illness are far from satisfactory. It is appropriate therefore to conclude this report by pointing out that these subjects offer great scope for research. Many can play a part in this work—paediatricians, obstetricians, anaesthetists, physiologists, social workers—indeed all those who are interested in the life and health of the next generation.

The following routine procedures might usefully be started without delay:

(1) The medical reports of all maternity hospitals should be standardised and should include comprehensive accounts of neonatal as well as of maternal results.

(2) Post mortem examination of babies born dead or dying in the neonatal period should become the rule, and accurate reports should be encouraged. The importance of this cannot be emphasised too strongly.

(3) There should be a general adoption of the accepted standards of immaturity and prematurity, with notification and follow-up of all cases.

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